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Table of Contents.

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ORIGINAL ARTICLES— Page.
Essential Enuresis: Successful Treatment Based on Physiological Concepts, by Neil D. Crosby .. 533
Tuberculosis at the Brisbane Children's Hospital: A Ten-Year Survey, by Felix Arden 543
The "Bad Risk" Anæsthetic in Emergency Surgery, by A. L. Nathan and R. A. Joske 545

REPORTS OF CASES—
A Case of Squamous Epithelioma of the Tongue in a Young Woman, Aged Nineteen Years, by Richard Flynn and H. Harris 548

REVIEWS—
The Medical Treatment of Gastro-Intestinal Disorders 550
Surgery for Nurses 550
Articular and Spinal Diseases 550
Emergency Surgery 550
Applied Medicine 550
Medicine and Mankind 551
A Text-Book of Obstetrics 551

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
The Royal Melbourne Hospital Medical Manual .. 551
Modern Treatment 551
Advances in Surgery 552
"Growing Girls" 552
Annual Meeting of the British Medical Association, 1949 552

BOOKS RECEIVED 552

LEADING ARTICLES—
The Treatment of Pulmonary Hydatid Disease .. 553

CURRENT COMMENT— Page.
Diphtheria Research in Germany 554
Dangerous Universal Donors 555
Twenty-Five Years of Medical Research 555

ABSTRACTS FROM MEDICAL LITERATURE—
Bacteriology and Immunology 556
Hygiene 557

BRITISH MEDICAL ASSOCIATION NEWS—
Scientific 558

CORRESPONDENCE—
Acropathy, Acrodynia Infantilis (Feer), Pink Disease (Swift) and Their Differential Diagnosis as to Ustilaginism (Mayerhofer) 562
Medical Practice in 1872 563

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 563

POST-GRADUATE WORK—
Auckland University College, University of New Zealand 564
Australian Society of Anæsthetists 564
The Melbourne Permanent Post-Graduate Committee 564

MEDICAL APPOINTMENTS 564

NOMINATIONS AND ELECTIONS 564

DIARY FOR THE MONTH 564

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 564

EDITORIAL NOTICES 564

ESSENTIAL ENURESIS: SUCCESSFUL TREATMENT BASED ON PHYSIOLOGICAL CONCEPTS.

By NEIL D. CROSBY,[1]

From the Department of Human Physiology and Pharmacology, University of Adelaide.

Introduction.

ENURESIS is one of the most harassing of medical problems which may affect a family. During childhood, it is exceeded in frequency of incidence by few conditions other than trauma and the acute infectious diseases. The considerable anxiety and labour which are caused by enuresis may be prolonged for years, so that these factors combine to make it an important part of pædiatric practice.

The magnitude of the problem is epitomized in the "Memorandum Prepared by the Joint Committee of the British Medical Association and Magistrates' Association on Psychiatry and the Law" (British Medical Association, 1948). Unfortunately, the conclusions of the committee do not offer much encouragement for sufferers.

This paper describes the author's concepts of the basic nature of the condition, the application of these concepts to a method of treatment, and the satisfactory results obtained by using this method alone, in over fifty consecutive refractory cases.

In a preliminary communication (Crosby, 1949*a*, 1949*b*) I gave a description of experiences in general practice which drew my attention to the natural history of enuresis. Medical practitioners have been so discouraged over the results of therapy in the past that there is now a widespread tendency for them to counsel parents "to wait and let the child grow out of it" when first consulted on the matter, or after an attempt at treatment with fluid restriction, alkalis and belladonna.

Most authors regard enuresis as a symptom rather than as a specific entity (Bakwin, 1949; MacGregor, 1948; Campbell, 1937; Winsbury-White, 1948). The varied opinions as to the ætiology may be divided according to four principal schools of thought which consider enuresis to be due to: (i) psychomotor activity, (ii) heredity, (iii) local urogenital defects or pathological conditions, and (iv) constitutional diseases.

To anticipate later discussion, I believe that the work described in this paper supports the view that in the great majority of enuretic patients, the removal of psychomotor activity is not necessary for cure. The results of this investigation, examined statistically, discredit hereditary and familial tendencies.[1]

Many therapeutic agents have been used in treatment. R. C. Browne and Ford-Smith (1941) have examined the effects of the drugs most commonly used for enuresis, in controlled experiments which did not show any one of

[1] Working at the University of Adelaide with a grant from the National Health and Medical Research Council.

[1] The following is a brief summary of the statistical analysis of the family histories of 73 patients. The incidence of enuresis in parents in this series is 17·8% at five years of age; this incidence rate is no higher than that assumed for the population (see incidence curve). There is no significant difference in the sex incidence in parents. The incidence rate in the siblings is 19·8% in this series; this is no higher than that assumed for the population. These two sets of facts taken together discredit any theory of inheritance factors and familial tendencies. The incidence rate in male siblings is not significantly greater than the incidence rate in female siblings.

these therapeutic agents to have specific value when compared with an inert control substance.

The figures for the incidence of enuresis will depend on the age limit beyond which it is first considered to be present and the completeness of the data. Thorne (1944) found an incidence of 16·1% at five years of age and of 8·5% at ten years among 1000 males who could make statements from their own memory. From this and other sources (British Medical Association, 1948; Bakwin, 1949; Browne and Ford-Smith, 1941; Blau, 1926; Sheldon, 1934), as well as my own series, an approximate age-incidence curve has been constructed for children who have wet beds.

The age incidence curve (Figure I) shows how unreliable it is to claim a cure when long periods of treatment are needed, in view of the continuously falling curve. In Thorne's series, only 10 out of 161 male persons who remembered having enuresis at five years of age regarded themselves as having been cured by therapy; the others considered that they "just grew out of it". More than

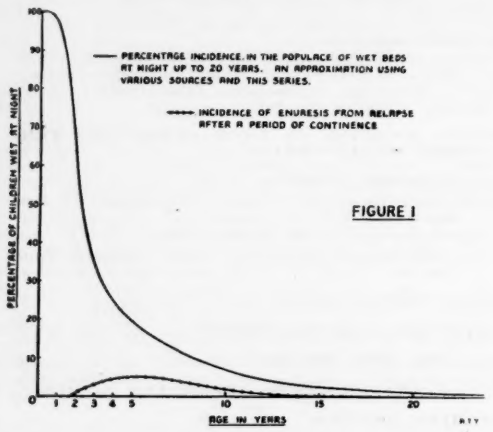

FIGURE I.

50% of the children who have enuresis at four years of age will be continent at ten years of age. For this reason any non-specific treatment can be instituted with occasional apparent success, especially if the treatment is prolonged over many months or even years, but not with repetitive results.

As treatments have been evolved, either empirically or from factors considered as ætiological, their failure in practice suggested the desirability of a fresh approach to the problem. Continued observations on patients and reflection on the results led to the concept that essential enuresis is not primarily a symptom, but that it is an entity or state arising from physiological activity, and is not of anatomical, pathological or psychological origin. On this basis a method of treatment was evolved, and its success in application is cogent support for the concept.

Definition of Essential Enuresis.

A definition must be in accordance with one's idea of ætiology. As an introduction, essential enuresis is defined as the involuntary and unconscious passing of urine after an arbitrary age limit of three years, in the absence of significant congenital or acquired defect or disease of the nervous and uro-genital systems, and in the absence of significant psychological defects. The discussion on ætiology will elaborate this view, which automatically excludes factitious enuresis (*vide infra*) and will allow an understanding of the many different aspects of the condition.

Descriptive Classification of Enuresis.

Enuresis is usually classified in accordance with observed facts as follows:

1. Nocturnal—that is, occurring at night. This is the commonest form.

2. Diurnal—that is, occurring by day only. This is relatively uncommon without concomitant nocturnal enuresis.

3. Continuous from infancy.

4. Reappearing after a period of continence.

5. Regular, with few if any dry nights unless some precautions are taken.

6. Sporadic, with varying intervals of continence.

7. Factitious—that is, a volitional act due to fear of the dark, dislike of the cold, or other reasons, so that the act is not really one of incontinence or enuresis.

Urinary Continence.

Micturition, like other essential functions, such as vasomotion, shows evidence of spinal cord control which has been superseded largely, and certainly directed, by higher

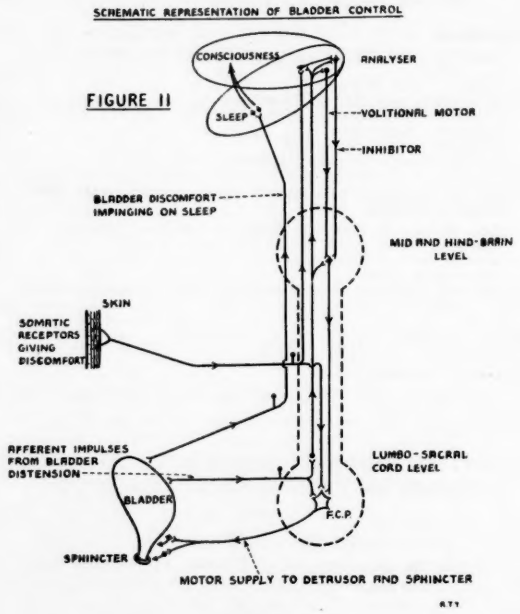

FIGURE II.

centres during progressive encephalization. The anatomy and physiology of micturition have been described accurately and at length by prominent authorities (Barrington, 1914; Brodal, 1948; Denny-Brown and Robertson, 1933; Goltz, 1874; Freusberg, 1874; Kuhn, 1949, Learmonth, 1931; Langworthy and Kolb, 1933; Neuman, 1949). Muellner and Fleischner (1949) have described the results of fluoroscopic examination during volitional micturition and inhibition and have concluded that the pubo-coccygeus muscle (voluntary striate muscle) is responsible for higher control, and that when this muscle in inactivated by a pathological condition or neural disease, automatic micturition only is possible. However, in view of the bladder control that some women retain after severe prolapse, and the prolonged inhibition that can enable the bladder to double its capacity after the first desire to urinate, it is difficult to believe that the detrusor is not under higher inhibition.

The normal stimulus for micturition is intravesical tension. A cystometrogram normally shows a rising type of curve on which serrations appear, depending on both bladder volume and rate of filling. At about 16 centimetres of water pressure a high spike heralds micturition. In adults the first desire to micturate is experienced at

about half the volume which will cause imperative urination (Best and Taylor, 1945). This physiological capacity is in turn about half the anatomical capacity of the bladder. The physiological values vary with the rate of bladder filling, the emotional state, cold and other factors. For instance, observations on a few children and adults show that in cold weather the volume of urine tolerated may show a reduction to half that which in hot weather produced the same acute desire to micturate.

Figure II is a schematic representation of the bladder control. The "centre" for the bladder is shown as a dumb-bell shape to take in the sacral cord and brain stem components. Afferent impulses arise from stimulation by a filling bladder and may result in three responses: (i) A cord reflex seen after spinal shock. This is not effective in completely emptying the bladder in the adult. (ii) A more coordinated reflex exhibited in decerebrate animals so that the bladder is emptied. (iii) A variable response in higher intact animals which are awake. Micturition may ensue or the animal may sustain inhibition for a longer period of time.

Usually the exercise of volition can initiate micturition at almost any time, irrespective of the degree of bladder filling.

Hemiplegia in adults often results in temporary or permanent incoordination of micturition, whereas hemiplegia from birth injury does not seem to interfere so much with the automatic bladder of infants. Thus, as in other functions, it appears that the higher centres progressively preponderate over lower ones in the phylogenetic and ontogenetic scale, and that the longer the higher control is exerted, the less likelihood there is of the lower centres reestablishing effective control. Likewise, the longer a conditioned response is maintained, the harder it is to extinguish it (Pavlov, 1927).

I believe that the study of micturition alone will not permit an understanding of urinary continence. For nocturnal continence two separate features are essential. Firstly, there must be inhibition of detrusor activity and micturition, so that micturition does not occur too early as a response to increasing bladder volume; this inhibition, together with the increasing anatomical capacity of the bladder as the child grows older, may allow the child to be continent all night without urinating if the volume of urine in the bladder does not reach the threshold value which is necessary to initiate micturition. More than this is necessary for continence, so that, secondly, this inhibition must remain prepotent over visceral afferent stimuli tending to initiate micturition until the discomfort of a filling bladder will disperse sleep, thus allowing the person to urinate voluntarily.

After all, in both continent and enuretic children, a time will come when the bladder contents reach the physiological limit. When this happens the continent child wakes, but the enuretic child does not. The experimental findings in this research suggest that enuretic children rarely have a physiologically full bladder at night.

The threshold volume necessary to wake the patient or to initiate micturition varies in both normal and enuretic children and adults. This is shown by the variation in the volume of urine which is passed by continent individuals when woken by the desire to urinate. This variation illustrates the reserve of inhibition which is available in cases of deep sleep due to fatigue, and represents one essential difference between the normal and the enuretic person. With long-established continence, the discomfort of a full bladder can amount to severe pain on waking from deep sleep.

The importance of these two factors, bladder inhibition and rousing from sleep, will be elaborated in relation to the present findings in enuresis.

Essential Enuresis: A Physiological State.

The age incidence curve of Figure I shows that all infants are incontinent from birth for a variable period of time. After the age of approximately eighteen months there is an interval when the labour associated with a "wet" child is regarded as some degree of nuisance, but the child is not regarded as abnormal until the age of about three years or more.

At the age of five years some 20% to 25% of children are still enuretic, and a complete reversal of opinion has occurred as to what constitutes a normal child with regard to urinary habits. Four-fifths of the children who as infants were "wet" and were of course considered normal, are continent at the age of five years, but the residual enuretic children are considered abnormal. If one narrows the gap too closely there comes a stage when the criterion for a normal child's urinary habits on one occasion becomes the diagnosis for enuresis on the next.

This leads to a consideration of the methods by which infants are converted to urinary continence, and requires more precise definitions of the state termed enuresis.

From the observations described in this paper, I have gradually developed these concepts: (i) that in most children essential enuresis is basically a physiological state, and does not involve as a primary cause anatomical defect, pathological condition or psychological aberration; (ii) that conversion from enuresis to continence usually occurs by physiological responses; (iii) that a further reversal from continence to enuresis is always possible.

Whether conversion can occur by natural means, or is acquired by artificial means alone, is a fundamental question. By "natural" I mean a physiological process which is inherent in the reaction of the organism to the environment. By "artificial" I mean those methods such as "potting", training, suggestion, hypnosis or volitional attempts to extend control during sleep, making continence dependent upon culture.

The Conversion Process for the Establishment of Continence.

If this approach is right, there should be evidence of the means by which the natural conversion process operates.

Sherrington (Denny-Brown, 1939) describes several reflexes operative in the spinal animal in relation to micturition and defæcation. Some of these reflexes tend to avoid soiling. Pinching the tail of a monkey after spinal transection in the mid-thoracic region sometimes interrupts micturition. "Micturition, if imminent when (spinal) defæcation sets in, is postponed" (Sherrington). Strong sensory stimuli will inhibit micturition in dogs which have had a dorsal transection of the cord (Goltz and Freusberg, 1874). These show that there are responses, inhibitory to micturition, organized at a spinal cord level, although I believe that in the human being continence largely involves "analyser" function. In any case, "analyser" activity is necessary for the establishment of conditioned responses or for reinforcement of them.

Observations on different mammals show that they are not prone to enuresis. This is not to be confused with so-called house training. From a teleological point of view, natural continence is an expected development, as a fouled animal is liable to leave a stronger spoor for his enemies and he runs a greater risk of bacterial infection. The continent animal has a material advantage for survival. In this connexion it is of interest that patients treated only for enuresis in this series have recovered spontaneously from meatal ulcers and mild vulvo-vaginitis after enuresis ceased, despite prior treatment for these conditions in the belief that they were causal factors.

If continence was artificially acquired and totally lacking in phylogenetical development, the faculty should show early dissolution under stress (Hughlings Jackson, 1932). In alcoholism, in narcosis for therapeutic reasons, and in recovery from anæsthesia it is very surprising how infrequently incontinence occurs. After extirpation of the anterior half of a dog's cerebral hemispheres the animal slept almost continuously, not even taking food; nor were sexual reflexes elicited, and yet the dog would wake just prior to defæcation and micturition. The dogs survived about a year (Pavlov, 1927). Bell and Karnosh (1949) describe the after effects of a right cerebral hemispherectomy in a man when bladder and bowel and sphincter control were unaffected.

Continence, dependent on volitional control, would not be expected in these conditions, especially in alcoholism in which there is often an inevitable diuresis, associated

with gross incoordination. Actually, urinary control is a very rugged mechanism when once continence is established.

In the human there is a clue to the conversion process in the discomfort evinced by an infant in the soiled state. I completely disagree with the statement: "It is plausibly said that an infant who cries when his nappies are wet is crying in apprehension of the disturbance of having them changed" (MacGregor, 1948).

I consider that the wet urinous state, which I term "somatic discomfort", acts as an inhibitory stimulus, and that this, at the onset of micturition, causes an unconditioned response tending to inhibit continuation of urination by inhibiting the detrusor and contracting the sphincter. This must not be interpreted in any way as a suggestion that micturition of itself leads to an inhibition of the act. It is the "somatic discomfort", consequent upon being soiled, which produces inhibition. Reinforcement of this inhibition from repeated soiling would be expected to produce a gradual increase in the tone of inhibition of the bladder, so that the volume of urine necessary to initiate urination would increase also. As the bladder fills, the inhibitory tone diminishes in prepotency over the increasing stimuli which travel over the afferent arm of the bladder reflex arc. At the same time, increasing bladder distension causes an increasing visceral afferent bombardment of the brain, which tends to impinge on and disperse sleep. In the sleeping child, then, the filling bladder is producing stimuli, which are active at two separate "centres", the bladder reflex centre and the "sleep centre", and the resultant effect is determined by three coexisting factors, depth of sleep, inhibitory tone and stimulation of bladder afferents. If the inhibitory tone is sufficiently prepotent, the discomfort of a filled bladder will disperse sleep before the bladder afferents initiate micturition as a reflex act. This process may begin very early, because young infants often remain dry until waking, immediately urinate and then cry from the discomfort of being wet.

The natural conversion process may be reinforced by some training methods, but more often there is a danger that these artificial methods will interfere with the natural process and even make the condition worse. There is evidence in support of this contention. Many children, of parents who have not tried to train the child, attain continence before acquiring speech. This is especially noticeable in some homes where the first child has had enuresis after much training; as a result, the parents have refrained from all efforts in view of the past dismal failure, and yet the siblings are continent from an early age.

The Mechanisms in Different Types of Essential Enuresis.[1]

I believe that the different types of essential enuresis are explicable in terms of concepts which are summarized below for reference. Some have already been elaborated.

1. The immediate "somatic discomfort" from being soiled by urine is a natural potent stimulus for the inhibition of further micturition. By repetition this leads to an increase in "tonic" detrusor inhibition.

2. For complete continence the inhibition of the detrusor must be sufficiently prepotent over the stimuli tending to cause micturition during sleep, so that the discomfort of a filling bladder will disperse sleep effectively before micturition occurs.

3. Conditioned responses to micturate can be acquired. These may interfere with or extinguish the "somatic discomfort" inhibition of detrusor activity.

4. Adaptation to many types of discomfort, including that of being soiled, can be rapidly attained, during sleep as well as when awake.

5. The inhibition developed by "somatic discomfort" is liable to diminution and extinction as a result of adaptation to discomfort as well as by acquirement of conditioned responses.

[1] The terms "response" and "reflex" are used interchangeably in this paper, "reflex" generally being used when reference is made to older papers.

The Simple Type of Essential Enuresis.

The simple type of essential enuresis is that in which the diurnal and nocturnal volumes of urine passed are relatively normal. The filled bladder initiates micturition before the discomfort of the distending bladder reaches the threshold to disturb sleep. These cases are seen when there have not been assiduous efforts at training which often results in totally undesired compulsive micturition. The simple type of enuresis is the only one to respond favourably to fluid restriction, as a result of which the bladder volume does not reach the threshold for micturition during sleep. A period of fluid restriction or hot weather, which causes sweating at night, may allow the establishment or restoration of a prepotent inhibitory tone; this is aided by a lessened depth of sleep consequent upon the removal of the repeated discomfort of being soiled. True continence may ensue as a result of this simple form of therapy.

The simple type is not common. The occasional equivalent of this type is seen in the wetting of the bed which occurs as a result of excessive tiredness in a normal child before the inhibitory reflex has been sufficiently long or deeply established. On such occasions the child usually wakes immediately after from the discomfort of being wet.

Many of the older children who have had the complicated type of enuresis when younger show evidence of reverting naturally to the simple type before becoming continent. The compulsive conditioned responses to micturate gradually diminish in potency and suffer extinction, leaving the patient with the simple form of enuresis—that is, where a full bladder will not wake him. The past history will show an alteration from regular nocturnal enuresis, in which small volumes of urine are passed, to irregular enuresis, with large volumes of urine either flooding the bed during sleep or being passed on the child's waking in the morning. These children often give a history that with fluid restriction, a quiet house and not going to bed too early, they will be dry without having to micturate during the night.

The Complicated Type of Essential Enuresis.

The simple type of enuresis is often complicated by superimposed conditioned responses which initiate micturition. Some of these are immediate responses, and some are well-established delayed responses.

Immediate Responses which may Initiate Micturition.—A common history is that it is almost impossible to go to the child during sleep without finding him urinating in sleep or just having completed the act. In these cases, light, noise, or physical disturbances from early training have been the conditioning stimuli which initiate micturition, so that this act is not related to physiological necessity or to a time interval after going to bed. Even doors slamming in the wind are enough to cause some children to micturate. Sometimes the history will reveal that there may be a dry bed when there has been a particularly quiet night in the whole house.

Delayed Responses which may Initiate Micturition after an Interval of Sleep.—Many infants are made comfortable at bedtime and then regularly disturbed after a certain time interval to be "potted". This is an ideal basis for the establishment of a delayed conditioned response which may even be repetitive. Is is especially dangerous if the child is not properly woken or if the procedure is repeated during the night. Some parents, in the hope of establishing continence, put the child to bed after a compulsory toilet, "pot" him again at the parents' bedtime, and then wake up at midnight to repeat the process. Careful recording of a urinary pattern, in the manner to be described, will show that some children micturate repetitively right through the night. The following (Table I) are the details of four consecutive nights, three from the "urinary pattern" and the fourth at the beginning of treatment in Case 41 (see treatment record, Table III). The results are highly suggestive of a delayed response. The history and "urinary pattern" of many patients suggest that the simple type is complicated by both immediate and delayed responses to micturate in sleep. Fluid restriction will not help such subjects.

TABLE I.

Night.	Bedtime.	Times and Volumes at Micturition.					
		Time (During Sleep.)	Volume. (Ounces.)	Time. (During Sleep.)	Volume. (Ounces.)	Time. (Waking.)	Volume. (Ounces.)
First	8.30 p.m.	11.25 p.m.	2	2.05 a.m.	1½	7.4 a.m.	1½
Second	8.30 p.m.	11.2 p.m.	1½	2.33 a.m.	1½	7.30 a.m.	2
Third	8.30 p.m.	11.17 p.m.	1½	2.04 a.m.	—[1]	7.30 a.m.	1½
Fourth	8.30 p.m.	11.30 p.m.	2	1.35 a.m.	1	7.15 a.m.	2½

[1] Bed wet: tube came loose during micturition.

Methods of Investigation and Assessment in this Series.

Most of these patients were referred to me by medical practitioners, and all had had previous treatment over varying periods of time. Histories and examinations were recorded on printed schedules, covering two foolscap pages. These could be amplified when necessary.

Special attention was given to the question of diurnal continence and day-time urinary habits, as far as these were known. An assessment of the child's mental age was made from both information and observation. It is necessary to gauge the intelligence of the patient.

The main points looked for in examination were obvious *spina bifida* indication, spasticity, trophic effects or awkward gait, and especially perineal anæsthesia. The urine was tested, particularly for albumin and sugar.

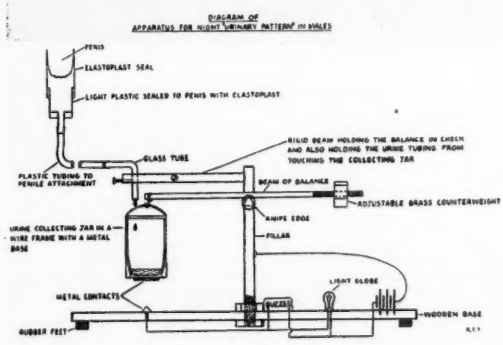
DIAGRAM OF
APPARATUS FOR NIGHT URINARY PATTERN IN BOYS
PENIS
ELASTOPLAST SEAL
LIGHT PLASTIC SEALED TO PENIS WITH ELASTOPLAST
RIGID BEAM HOLDING THE BALANCE IN CHECK AND ALSO HOLDING THE URINE TUBING FROM TOUCHING THE COLLECTING JAR
GLASS TUBE
PLASTIC TUBING TO PENILE ATTACHMENT
BEAM OF BALANCE
ADJUSTABLE BRASS COUNTERWEIGHT
KNIFE EDGE
PILLAR
URINE COLLECTING JAR IN A WIRE FRAME WITH A METAL BASE
LIGHT GLOBE
METAL CONTACTS
WOODEN BASE
RUBBER FEET

FIGURE III.

The child with normal diurnal control, and without signs of neurological disorder or gross uro-genital disease, was considered suitable for treatment on these grounds alone. The more regularly such a child wet the bed the better, provided, of course, that there was no reason to suspect factitious enuresis.

For at least three days and nights all the urine was collected from each patient. The times of micturition and the volumes of urine were recorded. This constituted a "urinary pattern" which was charted.

With female children, this necessitated waking the patient at intervals, but with male children a device was evolved which gave a close approximation to the natural pattern which occurred at night. The device was a simple knife-edge balance (Figure III). The counterweight allowed the sensitivity to be set so that any desired volume would cause the receiving jar to descend, thus making an electrical contact which caused a light or a buzzer, or both, to signal to the observer that micturition had occurred. The time of micturition and volume of urine were recorded and the jar was replaced without the child's being wakened, if possible. Usually the setting of the counterweight made the balance act after 10 to 15 millilitres of urine had collected. The observer then waited until micturition ceased. Taken in conjunction with the history, this "urinary pattern" enables the type of enuresis to be ascertained with reasonable certainty.

In therapeutic practice there is no need to delay treatment after completion of the history and examination, in order to obtain this "urinary pattern", unless there is reason to suspect some neurogenic disorder of micturition, which may be suggested by the history, or by undue diurnal frequency, or by precipitancy. When treatment is instituted, frequently from the first night and almost always after the first few nights, micturition is arrested after only a few millilitres or less of urine have been passed involuntarily. The amount of urine passed after waking is recorded on each occasion, and this automatically gives the night record or "urinary pattern". This can be augmented by the day record if desired. The majority of enuretics, however, can be treated without recourse to the diurnal pattern. Should the night volumes not show signs of reaching normal proportions, then the diurnal pattern should be estimated.

TABLE II.
Examples of Consecutive Night Readings.

Case Number.	Specific Gravity on Consecutive Nights.			
	First Night.	Second Night.	Third Night.	Fourth Night.
22	1022	1018	1008	1018
23	1010	1023	1023	—
35	1010	1026	1021	—
55	1007	1027	1026	—

In this series of cases, the urine was collected for three days and kept in twelve-hour portions, from 8.30 to 8.30. The constituent volumes of each twelve-hour period and the times at which they were passed were known. Each twelve-hour sample was then tested for pH, specific gravity, and chloride and urea concentration. This was done in this series to find how many children had nocturia and among other things what relation this bore to probability of cure (Freidell, 1927) and ætiology (Smellie, 1949; Cameron, 1927).

Results of Urine Estimations.

The estimations for chloride and urea concentrations were carried out in duplicate according to Schale's method and the hypobromite method respectively.

The urine estimations have been carried out at all months of the year. Only two patients have shown a night specific gravity of less than 1010, but these results were not repeated on other nights.

Examples of consecutive night readings with one low specific gravity are shown in Table II.

One girl (Case 48) gave night readings for specific gravity of 1014, 1011, 1010, with day readings of 1021, 1026, 1020; the total amounts of urine passed by day and by night were approximately equal. This child has done very well, and in the three months since treatment has not had a single wet bed.

One could not detect any relation to urinary acidity or alkalinity, the same child frequently showing variations on consecutive nights, or on "wet" nights, these variations seeming to depend on food and fluid intake and physical activity.

In enuretic and normal children, it is usually possible to identify the night urine from the day urine by the reversal of concentration of urea and chlorides. The day urea concentration is usually lower than the night, and the day chloride concentration is higher, frequently both reversals being present, although the chloride reversal is the more constant. Generally, there is an increased concentration of both chlorides and urea in the older children, in both day and night samples.

To summarize the effective results of these various estimations on urine: the "urinary pattern" was the only information found to be of positive value in relation to enuresis; no other results appeared to be significant as an indication of the ætiology or prognosis of enuresis.

The Apparatus Used for Treatment.

The method of treatment used in this series aims at extinguishing any conditioned responses which initiate micturition, and at reinforcing the natural method of building up the "inhibitory" tone, which is considered necessary for continence.

To practise the method it was necessary to devise a means of detecting the earliest onset of involuntary micturition, so that at this onset a variable stimulus could be applied. I hoped to avoid the perineal and genital area as the region for applying the mild stimulus, for obvious reasons, and fortunately the loin and lateral abdominal regions have been completely satisfactory sites for this purpose.

After much consideration, electrical means seemed the only practicable way for this to be done. Many technical difficulties were met and had to be overcome. The final unit proved satisfactory for clinical use in hospital or at home (see Figure IV).

The essentials of the unit are: (i) urine electrodes for detecting the onset of micturition, (ii) a sensitive relay, (iii) stimulating electrodes for applying an adjustable stimulus, (iv) a generator of signals to the observer and (v) a source of electromotive force.

There are separate male and female urinary electrodes, but the remainder of the apparatus is interchangeable. The electrodes must be kept electrically clean or preferably made of non-corrosive material.

The urinary electrodes consist of metal strips separated by a gap which is bridged by urine, surface tension sufficing to maintain contact. One drop of urine is sufficient to carry a current large enough to operate a sensitive relay. These urine electrodes must be adapted accurately to the patient and need to be easily changeable after micturition.

On micturition the relay operates circuits, which produce two effects: (i) Signals are made for the observer (parent or nurse) by both light and sound, so that the patient will be attended to. (ii) A stimulus, capable of variation to any desired intensity, is applied to the patient. The stimulating electrodes are carried by an elastic belt, fastened around the waist, and are applied to the loin region. The stimulus has to be varied for different patients and often during treatment. The doctor or parent can test the stimulus on the skin of his forearm. An intensity just less than enough to cause discomfort at this site is satisfactory for the first night. Subsequently, the intensity is varied as seems necessary depending on the reaction to the stimulus and the depth of sleep. The instrument is connected to the elastic belt by a four-wire cable; this is covered, so that it is resistant to acute bending—a safety measure to avoid entangling the sleeping patient.

Actual Treatment.

All these patients were treated by this method alone in order to assess the results accurately, although I shall point out later why adjuvant treatment is theoretically desirable in some selected cases.

Eight children and three young adults were treated in hospital or the treatment was begun in hospital. The remainder were treated at home by the parents, and this is desirable for several reasons. At home the child is in the environment to which he must be adapted, and the switch from hospital to home offers difficulty which fortunately does not seem so far to have had deleterious effects by causing a relapse. The parents are more likely to carry out instructions to the letter as they are vitally concerned. Unless an actual clinic is set up at a hospital there is the recurring necessity to instruct changing staff in the method. In a mixed ward there are always other duties which may take the nurse from the scene at the important times. Should the nurse have to leave the ward, it is essential that the instrument be switched off to avoid unnecessary discomfort to the child, especially if the child is young.[2]

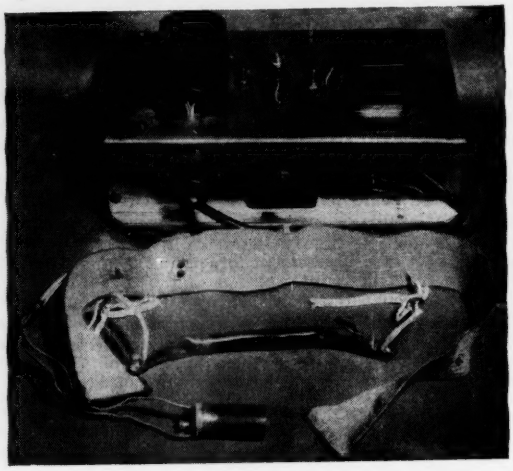

FIGURE IV.

Photograph of an instrument and the attachments used for treatment in this series.[1] The operative switch is on the front panel of the instrument, together with the stimulus adjustment and a four-wire plug. The right hand switch on the top is the test switch, which enables the unit to be tested and the strength of the stimulus to be adjusted prior to use on a patient. This particular instrument also has on the top a switch, which enables a variable thermorelay to be used, if it is desired to limit the time for which the stimulus can act, but it is not needed if the instructions are adhered to rigidly. The dial pointer is turned by small increments to adjust the strength of the inhibitory stimulus. In this photograph, the belt is connected by two leads to the male urine electrode, which is screwed into the plastic tube. The electrode and plastic tube are very light and are fastened to the penis with "Elastoplast" to make a loose but watertight seal. The female electrode is lying transversely and the quarter-inch wide elastic supports are fastened onto the loops of tape which are attached to the belt. The female patient wears this in the manner of a sanitary pad held firmly but not uncomfortably in the vulval cleft. The two stimulating electrodes are seen projecting from a small plastic rectangle on the belt. When the belt is fastened these make certain contact with the bare skin of the loin region. In practice the unit is placed on a chair at the head of the bed, and the cable is tied with tape to one of the bars at the head of the bed in order to prevent the unit's being dragged off by a restless patient. The cable is coiled loosely by the side of the patient.

The most important general instruction is that the observer must be within sight or hearing of the patient and instrument, and it is for this reason that the unit has been so constructed that there can be no stimulus without visual and auditory signals. When treatment is domiciliary it is essential that one parent must be sensitive enough to wake easily with the noise, otherwise it would be necessary for an attendant to remain awake. With

[1] The University of Adelaide has applied for a patent, in order to ensure that a satisfactory type of equipment will be produced, and is negotiating for the manufacture of suitable clinical instruments. Ample and illustrated instructions will be provided with these units.

[2] Some instruments had variable thermorelays, which limited the duration of the stimulus to five to fifteen seconds.

TABLE III.

Treatment Record: A Complete Record of Case 41.

Date.	Bed Time.	Hour Woken by Instrument.	Hour Woken Naturally.	Amount of Urine Passed. (Ounces.)	Remarks. Degree of Urinary Arrest, Reaction to Stimulus, Ease of Waking *et cetera*.
14/6/49	7.30 p.m.	11.30 p.m. 1.35 a.m.	 7.15 a.m.	2 1 2½	Immediate arrest. Immediate arrest. Immediate arrest. } Cried slightly on awaking. Woke immediately.
15/6/49	7.00 p.m.	9.45 p.m. 10.40 p.m. 1.20 a.m. 4.20 a.m.	 7.20 a.m.	3 1 2½ 2 1½	Immediate arrest. Immediate arrest. Immediate arrest. Immediate arrest. Immediate arrest. } Reaction as above except cried longer. Cut back stimulus from 110 to 90.
16/6/49	7.00 p.m.	1.00 a.m. 6.10 a.m. 7.15 a.m.		3½ 2½ 1	Immediate arrest. Immediate arrest. Immediate arrest. } Upset very little on waking.
17/6/49	7.30 p.m.	12.30 a.m. 7.00 a.m.		2½ 3½	Immediate arrest. Reaction as above.
18/6/49	7.00 p.m.	9.55 p.m.	 8.20 a.m.	1½ 7	Immediate arrest. Reaction as above.
19/6/49	7.05 p.m.	2.20 a.m. 7.15 a.m.		3¾ 3¼	Immediate arrest. Child found holding contacts away from his back with hand, when awoken.
20/6/49	7.15 p.m.	2.15 a.m.	 7.15 a.m.	5¾ 6	Immediate arrest. Not upset when waking. Wide awake when urinating.
21/6/49	7.15 p.m.		7.15 a.m.	6	Did not wake during night.
22/6/49	7.15 p.m.	11.00 p.m.	 7.25 a.m.	1 5	
23/6/49	7.15 p.m.		7.00 a.m.	6	Did not wake during night.
24/6/49	7.20 p.m.		7.10 a.m.	7	Did not wake during night.
25/6/49	7.00 p.m.		7.00 a.m.	6	Did not wake during night.
26/6/49	6.45 p.m.		7.15 a.m.	6	Did not wake during night.
27/6/49	6.45 p.m.		7.15 a.m.	7½	Did not wake during night.
28/6/49	6.45 p.m.	6.00 a.m.		8	Did not to to sleep again after being woken.
29/6/49	7.05 p.m.		2.00 a.m. 7.05 a.m.	9 10	Given approximately one and a half pints of water before going to bed. (Woke of own accord 2 a.m.) (Nine ounces.)
30/6/49	6.50 p.m.		7.15 a.m.	4½	Given approximately one pint of water before going to bed.
1/7/49	6.50 p.m.		7.15 a.m.	10½	Given approximately one and a half pints of water before going to bed.
2/7/49	6.50 p.m.		7.20 a.m.	8½	Machine not attached.

older patients who do not mind a more intense stimulus, it may not be necessary to have an attendant after the first night or two. Most children over eight years of age elect to do all the arrangements themselves, and are happy to sleep alone after the first few nights; usually the parents are instructed to leave the communicating doors open so as to be able to hear the auditory signal. If the patient is alone, then it is essential that the stimulus be sufficient to ensure waking, otherwise small points of electrolysis can appear at the stimulating electrodes and, of less importance, the batteries will run flat. A daily report to the doctor is desirable for the first few days in order that he may review results and give advice. The results of each night, including the volumes of urine passed and the effect of the stimulus, are recorded (see treatment record—Table III).

From the beginning the child is allowed to eat and drink as he pleases, to lead his usual life, and to practise his usual hour of retiring. As the child has been assessed from his history and examination, a diagnosis has already been made of the probable type of enuresis, and from this it will be known whether it is desirable to increase fluid intake at an early stage. Usually, one first sees an inhibition of the varied conditioned responses which are believed to initiate micturition. Treatment has to be prolonged until these responses are thought to be extinguished. Time alone will prove whether they have been permanently extinguished or whether circumstances will provide stimuli for the restoration of them. Later, increasing inhibition of the detrusor occurs, so that the child has greater intervals between urination and retains more urine in the bladder before being awakened by the electrical stimulus. At this stage the child may be dry all night and be continent on waking in the morning if he has not taken much fluid or if he has sweated considerably in the night owing to hot weather.

At this stage the child's enuresis has been converted to the simple type of enuresis, which will allow continence with treatment by fluid restriction. However, it still remains to be proven whether this inhibition of detrusor activity will be prepotent over the increasing potency of intravesical pressure which results from greater filling of the bladder. Such proof is obtained by increasing the fluid intake of the patient so that his bladder cannot hold a night's urine. He then experiences some more electrical stimuli as a rule. Some patients, however, exhibit complete inhibition after increasing the fluid intake at this stage, so that they do not experience further stimuli. As an arbitrary rule, treatment is discontinued after the patient has awakened naturally to micturate during five consecutive nights without any enuresis, this usually indicating cure.

Results of Treatment.

The establishment of continence is the only satisfactory criterion of successful treatment for enuresis, whereas "improvement" is an unsatisfactory state. Most of the methods hitherto practised for the treatment of enuresis recommend fluid restriction and perhaps lifting of the child at night, together with some special therapy. Consequently, the child may be reduced from nightly enuresis to two or more "wet" nights a week. By comparison with the past this can be called improvement, but it is more

TABLE IVA.

History of Enuresis Prior to Treatment and Result of Treatment. Class I; 29 Subjects, Aged Three and a Half to Ten and a Half Years, with Histories Suggesting Essential Enuresis.

Serial Number.	Case Number.	Age.		Sex.	Enuresis.			Treatment Started.	Result.
		Years.	Months.		From Birth.	Relapsed.	Also Diurnal.		
1	10	9	2	F.	Yes.			October, 1947.	Cured.
2	46	6	—	M.	Yes.			November, 1947.	Cured.
3	4	3	8	F.		Aged 3 years.		June, 1948.	Cured.
4	27	9	3	F.	Yes.			June, 1948.	Cured.
5	32	8	6	M.	Yes.			July, 1948.	Cured.[1]
6	47	3	10	M.	Yes.			August, 1948.	Cured.
7	16	5	1	F.	Yes.			August, 1948.	Cured.
8	39	5	—	M.	Yes.			November, 1948.	Cured.
9	20	6	11	F.	Yes.			February, 1949.	Cured.
10	44	8	6	F.	Yes.			February, 1949.	Cured.
11	12	4	1	M.	Yes.			March, 1949.	Cured.
12	18	8	11	F.	Yes.			March, 1949.	Cured.
13	36	9	—	F.	Yes.			March, 1949.	Cured.
14	15	10	4	M.	Yes.			March, 1949.	Cured.
15	13	9	8	M.	Yes.	Several times.		April, 1949.	Cured.
16	14*a*	6	5	M.	Dry at 2 years.	Aged 3 years after surgery.		April, 1949.	Cured.
17	14	10	3	M.	Dry at 2 years.	Aged 5 years after measles.		May, 1949.	Cured.
18	5	6	3	F.	Yes.			June, 1949.	Cured.
19	41	5	1	M.	Yes.			June, 1949.	Cured.
20	40	7	7	F.	Yes.			June, 1949.	Cured.
21	37	4	4	M.	Yes.			June, 1949.	Cured.
22	3	7	6	F.				June, 1949.	Cured.
23	6	7	6	M.	Yes.			July, 1949.	Cured.
24	23*a*	3	9	M.	Yes.			July, 1949.	Cured.
25	22	4	6	M.				July, 1949.	Cured.
26	11	10	5	M.	Intermittent till 7 years. Constant since.			July, 1949.	Cured.
27	50	5	10	M.	Yes.			August, 1949.	Cured.
28	8	6	10	M.	Dry at 2 years.	Aged 6 years after measles.		August, 1949.	Cured.
29	1	10	5	F.	Yes.			September, 1949.	Cured.

[1] This male child subsequently suffered relapse, but was not brought for further treatment. The late result is not known.

TABLE IVB.

History of Enuresis Prior to Treatment and Result of Treament. Class II; Six Subjects, Aged Three and a Half to Ten Years, with Histories Suggesting Either Incontinence or Essential Enuresis Complicated by Other Factors.

Serial Number.	Case. Number.	Age.		Sex.	Enuresis.			Treatment Started.	Result.
		Years.	Months.		From Birth.	Relapsed.	Also Diurnal.		
1	34	4	4	M.	Yes.		Yes.	June, 1949.	Cured.
2	28	5	6	F.	Yes.		Yes.	June, 1948.	Failure.
3	33	3	10	M.	Yes.		Yes.	June, 1948.	Cured.
4	51	4	1	M.	Yes.		Yes.		Nearly cured.
5	25	4	11	M.	Yes.				Cured.
6	52	10	3	M.		Aged 5 years.	Yes.		Cured.

apparent than real and has only reduced the labour of dealing with soiled linen without really ameliorating the other complications of enuresis. When the particular regime is abandoned through the lack of further progress, this "improvement" is rarely maintained.

An occasional lapse from continence is experienced by nearly all normal children in the more tender years. The history in Case 40 illustrates such incidents.

The mother, at the first interview, stated that her other two children "never wet the bed" and was quite sure on the point. The child with enuresis was referred by a consultant pædiatrician, and treatment was begun at the Adelaide Children's Hospital and completed at the child's home, complete continence ensuing in three and a half weeks. This girl had not a single lapse in the next six months, but the mother confessed that her other two children, each on a separate occasion, had wet the bed. She recollected then that there had been other rare examples in the past, which had been forgotten as they did not cause anxiety.

One can confidently state that no child, continent between three and eight years of age, has really been free from lapse of continence on at least one night. Consequently, no treatment could guarantee to prevent the occasional wet bed. Apart from this consideration, my present concept of essential enuresis as a physiological state is such that I expected that some patients would relapse as a result of conditions which might occur after treatment, but that such relapses should be readily amenable to a repetition of the treatment. These expectations were realized, and are discussed in the appropriate parts of the text.

Frequent reference is made in this paper to the increase in the bladder volume as a result of treatment. As all intraurethral manipulations were avoided in this series, there is no absolute check on this. If the increase was only apparent, owing to expulsion of a large amount of residual urine, one could have expected to find evidence of the distended bladder during preliminary examinations, more especially in those cases in which ten ounces or more would have to be postulated to explain the increase. However, all the evidence pointed to a real increase of bladder capacity.

The complete series of patients who have been treated by this method has been divided into three classes as follows.

Class I.—Children aged three and a half to ten and a half years without obvious neurogenic or uro-genital cause for enuresis. Twenty-eight patients were cured out of 29.

Class II.—Children aged three and a half to ten and a half years suspected of having neurogenic or other cause for enuresis or incontinence. Four patients were cured out of six. One result was a failure.

TABLE IVc.

History of Enuresis Prior to Treatment and Result of Treatment. Class III; 23 Subjects, Aged Over Ten and a Half Years.

Serial Number.	Case Number.	Age.		Sex.	Enuresis.			Treatment Started.	Result.
		Years.	Months.		From Birth.	Relapsed.	Intermittent.		
1	47*a*	13	3	M.	Dry at 2 years.	Aged 3 years.		August, 1948.	Cured.
2	42	12	—	M.	Yes.			September, 1948.	Cured.
3	38	13	—	M.	Yes.			September, 1948.	Cured.
4	19	28	—	F.	Dry at 2 years.	Aged 5½ years.	Yes.	September, 1948.	Cured.
5	2	14	—	M.	Yes.			November, 1948.	Cured.
6	7	11	10	M.	Yes.			January, 1949	Not cured.
7	17	17	1	F.	Yes			March, 1949.	Cured.
8	26	11	—	M.	Dry at 18 months.	Aged 5 years after burns and asthma.		February, 1949.	Cured.
9	24	17	6	F.	Dry at 4 years.	Aged 7 years after measles.		February, 1949.	Cured.
10	30	11	8	M.	Dry at 18 months.	Aged 3 years.	Yes at times.	October, 1948.	Cured.
11	35	21	5	F.	Yes.			December, 1948.	Cured.
12	21	12	7	M.	Yes.			April, 1949.	Cured.
13	45	17	7	M.	Dry at 16 months.	Aged 7 years.		June, 1949.	Almost cured.
14	43	13	8	F.	Yes.			June, 1949.	Cured.
15	48	11	2	F.	Yes.			June, 1949.	Cured.
16	23	12	5	M.	Yes.			July, 1949.	Cured.
17	29	11	9	M.	Yes.			July, 1949.	Cured.
18	9	11	1	M.	Yes.			July, 1949.	Cured.
19	53	38	3	M.	Yes.			September, 1949.	Cured.
20	49	11	10	M.	Yes.			September, 1949.	Probable failure.
21	54	24	—	F.	Yes.			November, 1949.	Cured.
22	73	24	7	F.	Yes.			November, 1949.	Probable failure.
23	72	13	2	M.	Yes.			November, 1949.	Cured.

Class III.—Children aged over ten and a half years without classification from history and examination. Eighteen patients were cured and there was only one outright failure in 23 patients.

Further Discussion in Relation to the Results of Treatment.

The belief that most children with enuresis pass excessive quantities of urine at night is widely held by parents and doctors. The "urinary pattern" shows that such is not the case. In this series, the night volumes were not abnormally large. A small amount of urine seems a great deal in a bed, especially when the child is found wet up to six times a night after the sheets and clothing have been changed. The results also showed that there was a considerable variation in the amount of urine passed by a patient in either the day or night twelve-hour period. The investigations and results have not supported the belief that nocturia is even relatively common (Smellie, 1949; Bakwin, 1949) or that a low specific gravity is associated with refractoriness to treatment (Friedell, 1927).

Pavlov found that the least number of stimuli necessary to establish a conditioned reflex was five and that most animals required between 10 and 40. It is significant that the least number of stimuli to produce continence, and that in one patient only, was five, and that the majority needed some 15 to 40 stimuli. Experimentally, a conditioned response, after extinction, is usually easily restored with fewer stimuli than were required for establishment, provided that there has not been long extinction and that there are no interfering factors, such as illness, change in environment or neurosis. Experiences with relapses of enuresis show parallel results. In enuresis the rate at which inhibition becomes prepotent seems to depend on the duration of the responses which initiate micturition, the length of adaptation to the wet state and the presence of nervous tension. Nervous tension always in experimental animals retards the development of conditioned responses, more especially inhibitory ones. It is of interest to note that Pavlov (1927) found that bromides and sedatives did not interfere with the establishment of inhibitory conditioned reflexes but did interfere with positive ones. Pavlov also found that neurosis extinguished conditioned responses and precluded the establishment of new responses, and that bromide aided the restoration to a conditioned state. Experimentally, conditioned responses cannot be established in some animals, the failure most probably being due to past experience. Some breeds of the same species are known to be more refractory than others.

The results obtained by this treatment are remarkably parallel with the classical experiments on conditioned responses.

There have been other attempts to use awakening methods after micturition has occurred (Pfaundler, 1904; Genouville, 1908; Remy-Roux, 1908-1911). The most notable is that of Mowrer and Mowrer (1938), who used a buzzer which operated when a pad on the bed was soaked with urine. These methods have been used either to see that the child is not left soiled or to superimpose a competitive positive conditioned response to wake at the time of micturition. The design of the present treatment differs radically, in that it is intended to build up bladder inhibition, so that the physiologically full bladder is the cause of the awakening; this is the fundamental requirement for continence in my opinion.

Any method relying on only one or very few stimuli is not likely to be following the path of a conditioned response, but is relying on psychological suggestion and the maintenance of volitional control during sleep. In the literature there are reports that enuresis has been cured after one or more subcutaneous injections of sterile water. It is notable that in this series there was no evidence of cure by suggestion. Considering the interview for a history, the investigation and the rather elaborate technique of treatment, one might have expected continence to be acquired forthwith, or within a day or two, by suggestion. Several of the patients were restless and woke of their own accord to micturate for one to three days from the beginning of treatment, but all relapsed, becoming adapted to the new circumstances. They then proceeded to develop continence in a manner which can be compared only with conditioned responses. Admittedly, most of these patients had been referred on by doctors after failure of treatment, or had sought advice because of such failure. Thus in over 50 consecutive patients no cure resulted which could be reasonably explained by suggestion.

Many factors are described by those holding that the cause of enuresis is pathological (Campbell, 1937; Whinsbury-White, 1948). In this series no attempt was made to treat such conditions. Nevertheless, no patients in Class I showed any sign of refractoriness on this score. One patient with a meatal ulcer and two patients with vulvovaginitis became spontaneously cured of the conditions after continence was established, although the three had been treated specifically for these conditions in the past

in the belief that the conditions were causal, not resultant. The urine rash, which is a common complication, clears up rapidly when once continence is established.

Although pathological and even neuro-muscular disturbances may exist in enuresis, they do not always preclude the possibility of continence being attained. Research work being continued at present shows that some such patients can become continent, but further work will be necessary to define the limits. A gross neurological lesion will, of course, lead to irremediable incontinence. The diurnal history is of great importance in these cases, for it may reveal diurnal enuresis, precipitancy and frequency associated with the passage of abnormally small volumes of urine.

Before institution of this method of treatment, a careful assessment of each patient is essential. Any congenital or acquired defects should be dealt with on their own merits and also so far as they affect the mechanisms described. I consider that it is impossible to assess any causal role of psychomotor activity in enuresis which has continued uninterruptedly from birth. I have not seen a relapse which appeared to be of direct psychomotor origin, but I concede that this is possible. I have insisted that psychological stresses are inimical to cure. Therefore, I believe that such stresses should be attended to, but only when there is indisputable evidence of them.

No other form of treatment was used in this series, in order to avoid confusion in assessment of results. That is why intraurethral and intravesical examinations were not made. However, I believe that adjuvant therapy is desirable at times in relation to the various mechanisms described. For nervous and excitable children, sedatives such as bromide and barbiturates are indicated. When sleep is so deep as to simulate narcosis, stimulants such as caffeine, ephedrine and "Benzedrine" should theoretically aid the conversion process. For the same reason a late afternoon sleep, by lessening the depth of nocturnal sleep, may assist in developing inhibition to the point where it will allow bladder discomfort to exceed the threshold necessary to cause wakening. Bladder sedatives and antispasmodics such as atropine will, in appropriate circumstances, lessen the potency of the bladder-emptying reflex.

Nash (1949) concludes that enuresis is a developmental error in 70% of cases, a view which is hardly compatible with an expectation of early cure in the majority of cases. The evidence of this series is that practically all subjects of essential enuresis can be cured in childhood by an application of the principles herein described. It also appears that after the age of ten years a cure is slightly less certain, for reasons which I hope have been adequately discussed in this paper, namely, that the complications of enuresis, especially those causing anxiety, become more prejudical to cure with increasing duration of enuresis.

Finally, it is strongly recommended that this method of treatment be first practised on some of the Class I children in order to gain experience of the different types of enuresis and the supervision to be adopted in the management of individual cases.

Summary and Conclusions.

1. Enuresis is a serious personal, family and social problem.

2. The views on ætiology are varied and conflicting.

3. Past methods of treatment are unsatisfactory.

4. The attainment of urinary continence is discussed. From this discussion a concept of the basic nature of essential enuresis is elaborated and defined on physiological principles.

5. Essential enuresis is divided into two primary forms, (i) simple and (ii) complicated. The factors which I believe lead to continuance of enuresis, or to a relapse after continence, are described on a physiological basis.

6. In this investigation, special attention was paid to the familial incidence and the "urinary pattern".

7. Heredity and psychological factors are discredited as primary factors in the ætiology of essential enuresis.

8. The method of treatment is based on the principles enunciated. By this method essential enuresis can be cured with near certainty. The certitude of cure diminishes somewhat after late childhood, because persistent enuresis produces complications which are prejudicial to recovery.

9. Cure is the only satisfactory criterion for successful treatment of enuresis. "Improvement" is misleading and disappointing.

10. Of 29 patients, classified as suffering from essential enuresis and aged less than ten and a half years, 28 were cured. The exception suffered relapse and was not again treated.

11. Of 23 patients over the age of ten and a half years and unclassified, 18 were cured.

12. The reason is given why fluid restriction is usually a failure in controlling enuresis. It is of real value only for the relatively uncommon simple enuresis. It is suggested that the failure of fluid restriction has helped to bring about a belief in a psychological ætiology.

13. There was no evidence that mild degrees of urogenital abnormality or of psychological stress prevent enuresis from being cured.

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Addendum.

Since the preparation of this paper, some further relevant information on treatment has become available.

Case 7 provides the only residual failure in Class III. The patient had been apparently cured and then relapsed into intermittent enuresis, subsequently becoming completely continent after some injections by his medical attendant. In Case 45 the patient has been continent for some months, after being treated for several relapses.

In Class I three more children have had relapses, two being easily cured again, while the third has not yet been again treated.

In the next ten cases all have been cured. The only protracted case was that of a boy, aged thirteen years, who had had enuresis severely and uninterruptedly, causing distress to himself and his family. He used the instrument for nearly three months at boarding school.

TUBERCULOSIS AT THE BRISBANE CHILDREN'S HOSPITAL: A TEN-YEAR SURVEY.

By FELIX ARDEN,
Brisbane.

IN a previous paper it has been shown that the Brisbane Children's Hospital admitted only 64 patients suffering from tuberculosis out of a total of 60,530 admissions during the ten-year period July 1, 1939, to June 30, 1949—a "tuberculosis rate" of 1·01 per thousand admissions. This compares favourably with "tuberculosis rates" (based on similar data), ranging from 3·81 to 10·39 per thousand admissions, for the principal children's hospitals in the other mainland States of Australia and is possibly one of the lowest in the world.

In this present paper an analysis has been made of the 64 Brisbane cases, so that the relative frequency of the different forms of the disease can be determined.

TABLE I.
Distribution of Tuberculous Lesions in 64 Children.

Types of Lesions.	Number of Cases.
Single lesions—	
Tuberculosis of cervical glands	30
Tuberculosis of mediastinal glands	2
Tuberculosis of spine	4
Tuberculosis of hip joint	5
Tuberculosis of knee joint	1
Tuberculosis of ankle joint	1
Tuberculosis of pleura	2
Tuberculosis of meninges	5
Total	50
Multiple lesions without miliary spread—	
Tuberculosis of spine and cervical glands	1
Tuberculosis of spine and mediastinal glands	1
Tuberculosis of spine and lungs	1
Tuberculosis of tibia and lungs	1
Tuberculosis of hip and kidney	1
Total	5
Single or multiple lesions with terminal miliary tuberculosis—	
Tuberculosis of lymph glands (generalized)	1
Tuberculosis of mediastinal glands and pleura	1
Tuberculosis of cervical glands, bowel and meninges	1
Tuberculosis of mesenteric glands, bowel and meninges	1
Tuberculosis of bowel	2
Tuberculosis of bowel and lungs	1
Tuberculosis of meninges	2
Total	9

The general distribution of the tuberculous lesions is set out in Table I.

The principal comment to be made on Table I concerns the absence of many common forms of tuberculosis. There have been, over a ten-year period at the Brisbane Children's Hospital, no children with uncomplicated tuberculous lesions of the lung, kidney, bowel, peritoneum or mesenteric glands. Tuberculous peritonitis has not been seen at all, and tuberculosis of the lung, kidney and bowel has occurred only in conjunction with other lesions.

In the succeeding paragraphs the tuberculous lesions in the 64 children under review have been classified and are described under the systems affected.

Tuberculosis of the Lymphatic System.

Uncomplicated cervical gland tuberculosis was found in 30 children during the ten-year period and accounted for practically half the total cases. The average age of the children at the time of admission to hospital was five

years. All of them were white children. There were no deaths in this group.

The diagnosis was established by histological examination of an excised gland in 24 cases. Tuberculosis of the tonsils was demonstrated in one of the remaining cases and in another tubercle bacilli were found in caseous material aspirated from a suppurating gland. Diagnosis in the remaining four cases was made on clinical grounds, supported by a positive response to the Mantoux test.

Unfortunately steps were not taken to determine whether these cervical gland infections were of human or bovine origin. It is suggestive that only two of the 30 patients gave a family history of tuberculosis, whereas five children were recorded as drinking unboiled milk, and many more drinkers of raw milk might have been found by close questioning. One child—a drinker of unboiled milk—was proved to have a bovine infection. During the period

TABLE II.

Site and Nature of Tuberculous Lesion.	Number of Cases.
Cervical glands only	30
Cervical glands and spine	1
Mediastinal glands only	2
Mediastinal glands and spine	1
Mediastinal glands and pleura, with miliary spread	1
Cervical glands and bowel, with miliary spread	1
Mesenteric glands and bowel, with miliary spread	1
Generalized tuberculosis of lymph glands	1
Total	38

under review only about 50% to 75% of milk consumed in the metropolitan area was pasteurized; the remainder, sold as "warm milk", came in many cases from untested herds, and tubercle bacilli were not infrequently found in random samples. It has always been a matter for surprise how seldom the consumption of tuberculous milk in Brisbane has caused clinical tuberculosis.

Treatment was by excision of the larger affected glands in 24 cases, but no attempt at block dissection of the neck was made. Two children had their caseating glands incised in mistake for a pyogenic abscess. These healed slowly. Two others were treated by aspiration alone, and in the remaining two the enlargement of the glands subsided without active treatment.

In only one child was tuberculosis of the cervical lymph glands accompanied by a tuberculous lesion elsewhere in the body. This child, a boy, aged three years, had a proved bovine infection and tuberculosis of the thoracic portion of the spine.

As has been previously stated, no child was found to be suffering from uncomplicated tuberculosis of the mesenteric glands—a point to be borne in mind when the differential diagnosis of abdominal pain is considered.

One child has been classified as suffering from generalized tuberculosis of lymph glands, a rare form of the disease, which will be the subject of a separate case report.

Tuberculosis of Bones and Joints.

As usual, tuberculosis of the spine and of the hip was much more common than that of other bones and joints. Out of 16 affected children, seven had involvement of the spine and six had a lesion of the hip joint.

According to our figures, one child in four who contracts tuberculosis sustains a bone or joint lesion. However, the prognosis is good. No child developed a miliary spread of his disease, and the only death among these 16 children was that of an aboriginal boy, aged six years, with uncomplicated tuberculosis of the spine, who collapsed and died under an anæsthetic during the application of a plaster cast to his body.

Treatment of the children with spinal lesions was by bone grafting after preliminary rest, and of those with affected hips by prolonged immobilization. Quiescence was eventually achieved in every instance.

TABLE III.

Site of Tuberculous Lesion.	Number of Cases.
Spine only	4
Hip joint only	5
Knee joint only	1
Ankle joint only	1
Spine and cervical glands	1
Spine and mediastinal glands	1
Spine and lungs	1
Tibia and lungs	1
Hip and kidney	1
Total	16

Tuberculosis of the Respiratory Tract.

Six cases of respiratory tract tuberculosis among 60,000 hospital in-patients is an incredibly small number and presumably indicates that children in this part of the world stand little chance of acquiring air-borne infection. Actually only three children (apart from those with miliary tuberculosis) had pulmonary involvement demonstrated radiologically or at autopsy. The details are as follows.

CASE I.—T.D., a white male child, was aged ten years. The mother has tuberculosis and his father died of the same disease. The child had caries of the eleventh and twelfth dorsal vertebræ. X-ray examination revealed "bilateral pulmonary infiltration". No tubercle bacilli were detected in the sputum. Progress X-ray films revealed gradual disappearance of the lung shadows.

CASE II.—R.G., a white male child, aged thirteen months, had no family history of tuberculosis. He was suffering from tuberculous osteitis of the tibia. Tubercle bacilli were isolated from the bone and from stomach washings. An X-ray examination revealed "diffuse consolidation in the right upper and middle zones consistent with a primary tuberculous infection". The child was removed from hospital at the parents' risk and was subsequently untraced.

TABLE IV.

Site and Nature of Tuberculous Lesion.	Number of Cases.
Pleura, with effusion	2
Pleura, mediastinal glands with miliary spread	1
Lung and bone	2
Lung and bowel with miliary spread	1
Total	6

CASE III.—D.F., a white male child, aged nineteen months, was admitted to hospital with a short history of fever and cough and died four days later. Autopsy revealed a large area of caseous bronchopneumonia in the upper lobe of the right lung, together with extensive tuberculous enteritis and terminal miliary spread.

It is noteworthy that no cases of the adult or reinfection type of apical tuberculosis with cavitation were encountered in this series.

Tuberculosis of the Gastro-Intestinal Tract.

With the exclusion of one child (R.G., Case II), examination of whose stomach washings revealed tubercle bacilli but who was otherwise symptom-free, there were five cases of tuberculous enteritis in the series. In each instance the bowel lesion consisted of multiple tuberculous ulcers, and had been accompanied by generalized and fatal miliary

spread. In fact, tuberculous enteritis was a post-mortem finding only and undiagnosed during life.

This compels the thought that a milder and reversible type of tuberculous enteritis might have been found occasionally if carefully sought. However, no children with tuberculous peritonitis were seen, no cases of ascites or intestinal obstruction were found due to tuberculosis, and there were no accidental radiological discoveries of calcified mesenteric glands. This is rather strange when one considers the relative frequency of cervical gland tuberculosis and the risk of consuming contaminated milk.

Renal Tract Tuberculosis.

Apart from children on whose kidneys at autopsy miliary tubercles were found, the case records reveal only one who could be said to have had renal tract tuberculosis. This patient was a white boy, aged four years, with tuberculosis of the hip joint, who upon one occasion had an attack of hæmaturia. Tubercle bacilli were grown from the urine. He soon recovered without further evidence of renal damage and remained well.

Tuberculosis of the Central Nervous System.

In this series of 64 cases of tuberculosis there were five children who suffered from tuberculous meningitis as an uncomplicated lesion, and four others in whom the meningeal involvement was associated with a generalized miliary spread. Each of these children died, streptomycin being unavailing in the one case in which it was used. It will be evident that tuberculous meningitis, which occurs only on an average of once every two years, is an uncommon form of meningitis at the Brisbane Children's Hospital. At least four varieties of pyogenic meningitis—influenzal, meningococcal, streptococcal and pneumococcal—are seen more frequently.

Mortality.

There were 15 deaths from tuberculosis out of 64 cases during the period under review. Five children died of tuberculous meningitis and nine of miliary tuberculosis. The other suffered from tuberculosis of the spine and died under anæsthesia while having a plaster cast changed. Post-mortem examination was made upon 12 of the deceased children.

If the 30 children with cervical adenitis are excluded from consideration, the death rate rises to 15 out of 34 cases of all other types of tuberculosis. This is a very high figure, and it indicates that childhood tuberculosis, once acquired, is still an extremely dangerous disease, even under the most favourable conditions.

Tuberculosis in Aborigines.

Eight of the 64 children affected with tuberculosis were wholly or partly of aboriginal descent. It is not possible to secure accurate figures concerning the proportion of aboriginal to white children in Queensland. Figures supplied by the Director of Native Affairs indicate a mainland aboriginal population, including half-bloods, of 15,857 under the control of his department, with perhaps another 1000 not so controlled, a total which represents about 1·4% of the entire population. The greater proportion of the aborigines live in the extreme northern and western areas of the State and would be unlikely, if ill, to seek admission to a hospital so far away as Brisbane. It is reasonable to assume that the proportion of aboriginal to white children in the "public hospital" population in the Brisbane area is well below 1%. However, aborigines account for 12·5% of all cases of childhood tuberculosis, and (if tuberculous cervical adenitis is excluded) for no less than 23·5% of the more severe types.

It is hard to explain the apparent immunity of aboriginal children to tuberculosis of the cervical glands.

The figures are not large enough to give an accurate indication of the relative tuberculosis mortality rates for aboriginal and white children. Four out of eight aboriginal children died, and (tuberculous cervical adenitis excluded) 11 out of 26 white children. The difference is not significant.

Summary.

A survey has been made of all tuberculosis patients admitted to the Brisbane Children's Hospital over a ten-year period. There were 64 such cases out of 60,530 admissions. The survey revealed no uncomplicated tuberculous lesions of the lung, kidney, bowel, peritoneum or mesenteric glands. Only three children (apart from those with miliary tuberculosis) had pulmonary lesions demonstrated radiographically or at autopsy. No examples of the adult or reinfection type of apical tuberculosis with cavitation were seen. Tuberculosis is relatively more common in aboriginal than in white children.

THE "BAD RISK" ANÆSTHETIC IN EMERGENCY SURGERY.[1]

By A. L. NATHAN,

Senior Resident Anæsthetist, The Royal Melbourne Hospital, Melbourne,

AND

R. A. JOSKE,

Senior Resident Resuscitation Officer, The Royal Melbourne Hospital, Melbourne.

THE problem of the "bad risk" anæsthetic is encountered in all hospitals dealing with emergency surgery, and no really satisfactory solution of this problem has yet been found. This paper discusses the present methods employed and the lessons learnt in dealing with anæsthetic and resuscitation problems in the management of emergency surgical cases in a large general hospital with a capacity of 560 beds. It is our experience that the hazards in the management of these cases can be minimized by the active cooperation of the whole team involved—surgeon, anæsthetist and resuscitation officer. It is the province of the two last-mentioned to prepare the patient for operation in as short a time as possible, and to ensure that adequate blood and fluid replacement is carried out. They are also responsible for the maintenance and recording of the patient's general condition before, during and after operation; and for the provision of anæsthesia adequate in both type and duration for such surgical procedures as are necessary.

The methods described are based on a study of some 500 patients whom we have ourselves treated. The cases include all types of conditions except pædiatric and obstetrical conditions; patients in these classes are rarely admitted to this hospital.

CLASSIFICATION.

The cases have been classified according to the condition producing the main anæsthetic danger. The basis of classification is anæsthetic assessment and not the primary surgical diagnosis. There are six main classes, as follows:

1. Shock-hæmorrhage syndrome: (*a*) Characterized by blood loss, either before or during operation. (*b*) Shock without gross blood loss.
2. Vomiting.
3. Poor cardio-respiratory reserve.
4. Electrolyte depletion and dehydration.
5. Thoracic emergencies.
6. Head injuries.

These classes will now be discussed in more detail. Although they are considered separately, most patients present more than one of these conditions, and no complete physiological separation is possible; for example, in a case of hæmatemesis the effects of shock and hæmorrhage and the danger of vomiting during induction of anæsthesia may be present.

[1] This paper was presented in part at a meeting of the Australian Society of Anæsthetists (British Medical Association), Victorian Branch, on May 19, 1950, at Melbourne.

MANAGEMENT.

The management of the six classes of emergency will now be considered.

The Shock-Hæmorrhage Syndrome.

Shock and Hæmorrhage with Massive Blood Loss.

Emergencies in the class covered by the shock-hæmorrhage syndrome with massive blood loss include ruptured spleen or liver, ruptured ectopic pregnancy, emergency gastrectomy for hæmatemesis and blood loss from external wounds.

Blood loss increases the anæsthetic risk because of the depletion of circulatory volume and rapid production of anæmia, which in turn decrease the cardiac reserve and may precipitate severe surgical shock and circulatory failure even in young persons. Any anoxia in these patients is thus of added significance, since the normal compensatory mechanisms are already in action. Included in this class are cases in which severe hæmorrhage is expected during the operation.

In dealing with these cases, emphasis must lie on adequate blood replacement before and during the operation.

No clear-cut indications for blood transfusion can be given, but it is generally indicated if the pulse rate is greater than 120 per minute, the systolic blood pressure less than 80 millimetres of mercury, or the hæmoglobin concentration less than 50% (100% = 14·6 grammes per 100 millilitres). Blood transfusion is mandatory if air hunger is present. These figures may be modified if, for example, there is previous hypertension or anæmia of long duration. The blood pressure is the most reliable indication for blood transfusion because of the occasional occurrence of brachycardia of vagal origin in patients suffering from severe shock.

Transfusions in this series have been given by the intravenous route, intraarterial transfusions not having been used. Although it is difficult to judge from the small number of cases presented by Robertson *et alii* (1948), it would not appear that intraarterial transfusion has any great advantage over transfusion given by the intravenous route. The transfusion must be given at a pressure greater than the normal systolic blood pressure, and hence blood must be pumped rather than allowed to flow under gravity; thus more elaborate apparatus and technique are required. The danger of arterial obstruction with local gangrene is incurred and the number of suitable arteries is much less than the number of superficial veins in any patient. On the other hand, smaller amounts of blood are needed and the transfusion may be given more slowly. Intraarterial transfusion is stated (Robertson *et alii*, 1948) to be more physiological than intravenous transfusion, as the blood pressure is raised by direct increase in the amount of blood in the arterial side of the circulation. However, blood given intravenously increases cardiac output by ensuring an adequate venous return (the primary deficiency in cases of hæmorrhage), and in addition is oxygenated by passage through the pulmonary circuit before distribution to the peripheral tissues. In our opinion, therefore, the advantages claimed for the intraarterial method of transfusion are outweighed by the additional danger incurred and the more elaborate apparatus and techniques required.

If blood is not immediately available, the patient's blood group and Rh type must be determined and he must be sent to the operating theatre with an intravenous saline or serum drip transfusion running. Blood may be given rapidly in most cases, Tovey (1950) recommending an initial rate of one pint in five to ten minutes. During the operation the rate must at least equal the rate of blood loss from the operative site; but if there is much operative shock, larger amounts will be needed.

The pre-operative use of morphine varies. It is indicated when severe pain is present, or when much handling of the patient is unavoidable (for example, in radiological examination), and it is also of value in the treatment of surgical shock. It must be remembered, however, that morphine is a powerful respiratory depressant, and should be used in minimal effective doses not less than half an hour prior to anæsthesia. If morphine is used, an ampoule of nikethamide (two millilitres of a 25% solution) should also be given to lessen the respiratory depression.

With adequate blood replacement, the choice of anæsthetic is dictated by other considerations as discussed below. The most important point in anæsthetic technique is adequate oxygenation. At no time during the induction or maintenance of anæsthesia should the patient be allowed to become anoxic, since the addition of anoxic to anæmic anoxia may precipitate severe and possibly irreversible shock. Unless intubation and controlled respiration are to be instituted, thiopentone should be avoided if possible, because of its production of central respiratory depression, except in minimal "sleeping" doses. The most satisfactory anæsthetic that we have found is a closed-circuit ether administration following cyclopropane induction, which enables high oxygen concentrations to be maintained. Trueta (1944), in a large series of cases under war conditions, found that ether was of benefit in dealing with patients suffering from shock, although this was before the introduction of thiopentone-curare anæsthesia. We have found that both these types of anæsthesia can be combined, with better results.

At the conclusion of the anæsthetic it is important to make a gradual change to mixtures of a lower oxygen concentration. Post-operatively, oxygen is administered by nasal catheter, "B.L.B." mask or oxygen tent, the choice depending upon the oxygen concentration required, the cooperation of the patient and the amount of nursing attention needed. Blood transfusion is continued until the patient's pulse rate and blood pressure are stable and a minimum hæmoglobin value of 80% is attained.

Shock without Gross Blood Loss.

Shock without gross blood loss is found chiefly in traumatic cases and in cases of recently perforated peptic ulcer.

These patients are treated as in the manner described for shock with gross blood loss. However, when there is shock without gross blood loss, there is considerable hæmoconcentration with hyperproteinæmia. Therefore, intravenous saline infusions are administered, either alone or with smaller amounts of blood or serum or both. It must be remembered that patients suffering from shock are more sensitive to the action of drugs and anæsthetic agents than normal persons, and consequently smaller dosages than normal must be used. Further, in the presence of peripheral circulatory failure, drugs given hypodermically are largely unabsorbed until the circulation is restored, when the normal rate of absorption causes sudden delayed action of the drug. For this reason morphine, atropine *et cetera* should always be given intravenously to ensure immediate action with no delay in absorption or accumulation of the drug at the site of injection.

Vomiting.

Emergency patients are especially prone to vomit during the induction of anæsthesia. They fall into two main groups: (*a*) those with intraabdominal lesions, especially intestinal obstruction; (*b*) those who have had a meal shortly before sustaining an injury requiring operative treatment.

The stomach must be emptied as completely as possible by means of a stomach tube before the operation is commenced. The patient is taken to the operating theatre with a Rehfuss's or Ryle's tube passed into the stomach via the nose. Induction of anæsthesia is performed in the operating theatre with the shoulder pieces of the operating table in place so that the Trendelenburg position may be assumed rapidly should it become necessary. An efficient suction apparatus in full working order must be beside the anæsthetist.

Because of its rapidity of action, the inductive agent of choice is "Pentothal" with curare, followed by the immediate passage of a previously tested, cuffed endotracheal tube. In order to ensure full oxygenation during the period from the administration of the "Pentothal" to the passing of the endotracheal tube, an assistant should

hold to the patient's face during this interval the facepiece connected to the oxygen supply from the anæsthetic machine. It must be remembered that curare releases the sphincteric action of the diaphragm on the cardia, allowing, in the prone position, regurgitation of gastric contents, especially with raised intraabdominal pressure in cases of intestinal obstruction and severe abdominal distension. Should vomiting or regurgitation of gastric content occur, the patient should immediately be placed in Trendelenburg's position and the naso-pharynx cleared by suction. A metal sucker must be used, as extensive experience has shown that the suction obtained with a soft rubber catheter is inefficient.

During the operation continuous or intermittent aspiration of the stomach is advisable, as further regurgitation may result from operative manipulation and pressure on the abdomen.

At the conclusion of the operation, when the cough reflex has returned, any residual debris should be removed from the mouth, the trachea aspirated with a soft sterile catheter via the endotracheal tube to ensure that there has been no leakage around the cuff, the cuff deflated and the endotracheal tube removed. The patient is returned to the ward in the lateral position.

If inhalation of vomitus has occurred, immediate post-operative bronchoscopic aspiration must be instituted.

Poor Cardio-Respiratory Reserve.

Poor cardio-respiratory reserve is found in conditions such as the following: cardiac failure from any cause —for example, valvular disease, hypertension, chronic nephritis; angina of effort; chronic lung disease such as emphysema, tuberculosis or bronchiectasis; gross arterial disease without hypertension; long-standing anæmia in which rapid blood transfusion is contraindicated. Hence it is especially important in old persons and those suffering from malnutrition—for example, thiamine deficiency in chronic alcoholics. In previously healthy young persons with severe shock or blood loss, the cardio-respiratory reserve is correspondingly diminished (*vide supra*).

Assessment of such patients may be difficult under emergency conditions. When possible a history of their "normal" exercise tolerance is obtained, with special reference to cough and sputum, previous episodes of cardiac failure, or chest pain or dyspnœa related to exertion.

In such cases emphasis must again lie upon full and adequate oxygenation during anæsthesia. In our experience, we have found that despite varied opinions thiopentone induction of anæsthesia is of benefit to the patient, both psychologically and physically. This has been questioned, but we consider that the advantages counter the disadvantages. The respiratory depression and fall of blood pressure which may occur can be offset by the adequate administration of oxygen by nasal catheter or facepiece connected to the anæsthetic machine prior to and during induction. The drop in blood pressure is usually slight, but in hypertensive patients it may be large—for example, up to 50 millimetres of mercury. The great advantage of this technique is that it eliminates or reduces to a minimum the struggling that so frequently occurs with other types of induction. This struggling in itself can precipitate cardiac failure in patients with an emergency condition and previously poor cardiac reserve. The amount of thiopentone given should be minimal—just sufficient to induce sleep. Less than 0·25 gramme suffices in most cases.

Maintenance of anæsthesia is best conducted with ether, preferably through a closed circuit because of the high oxygen concentration that can be obtained. It is important that at the conclusion of the anæsthetic a high oxygen concentration be maintained by nasal catheter, "B.L.B." mask or oxygen tent. Davies and Mackinnon (1949) have recently stated that high oxygen concentrations are dangerous in cases of chronic *cor pulmonale*. These findings have not been confirmed, but oxygen should be given cautiously in such cases. Cyclopropane should not be used for patients with cardiac arrhythmia because of the difficulty in gauging the depth of anæsthesia in such conditions.

Intravenous therapy in these cases must be conservative. Rapid fluid replacement is contraindicated, as a rapid increase in blood volume may precipitate cardiac failure and pulmonary œdema. Fluids containing sodium chloride should be used sparingly, as sodium ion retention with consequent water retention has been shown to be an important factor in cardiac œdema (Stead, 1948). Should pulmonary œdema occur, venesection is the only efficient immediate treatment. "Digoxin" (0·5 to 1·0 milligramme) should be given intravenously, and "Neptal" is indicated in the absence of known renal disease or gross albuminuria.

Pneumonia *per se* is not a contraindication to intravenous fluid and electrolyte replacement, as patients with fever and increased metabolism have increased fluid and salt loss, and hence require increased replacement. In cases in which anæmia complicates lung disease, blood transfusion may be needed to overcome deficient pulmonary aeration. If the cardio-respiratory reserve is low, packed cells should be used.

Electrolyte Depletion and Dehydration.

The most potent cause of peripheral circulatory failure is electrolyte depletion and dehydration resulting from lesions such as pyloric stenosis, late intestinal obstruction and general peritonitis. As far as possible these losses should be made good by rapid intravenous infusions before and during operation. Until the blood pressure reaches a satisfactory level, saline should be given by the intravenous route as fast as is possible without evidence of circulatory overloading—namely, increased cervical venous pressure, tachycardia, basal crepitations, dyspnœa and pulmonary congestion. In severe cases blood or serum may be needed, but the former is undesirable when there is marked hæmoconcentration unless this is associated with acute blood loss. The patient's condition should be maintained by a slow intravenous infusion of normal saline unless the blood pressure again falls, when saline, serum or blood should be administered rapidly. The choice and amount of fluid given are determined by clinical assessment—pulse rate, blood pressure, urinary output and reaction, specific gravity and chloride content of the urine. It should be noted that estimations of urinary chloride content (Fantus test) are unreliable when there has been loss of sodium ion in excess of chloride ion. Denton (1949) has shown that renal excretion of chloride ion is determined, not by plasma chloride levels, but by the sodium to chloride ratio, or the pH of the extracellular fluid, or both. We have confirmed this observation in four cases. Hæmoconcentration is gauged by hæmoglobin value (Sahli method) and serum protein estimation (copper sulphate method).

Pre-operative therapy in these cases is of the utmost importance in ensuring adequate circulatory volume and in eliminating or at least lessening the risk of peripheral circulatory failure, thus ensuring adequate tissue oxygenation throughout the operative and post-operative periods.

Post-operatively, intravenous therapy should be continued until the patient can maintain his fluid and electrolyte balance by the oral or rectal route, or by a combination of both. The time taken for this to occur varies with the condition of the patient when first examined, the operative procedure undertaken, and the site, extent and progress of the causal lesion. The amount and type of fluids required vary within wide limits, being determined by the criteria given above for initial assessment, accurate recording of fluid losses, and laboratory determination of the extracellular electrolyte pattern.

Thoracic Emergencies.

Thoracic emergencies fall broadly into traumatic and non-traumatic groups; but both types present similar problems and will be considered together. The important factors are sudden decrease in vital capacity, intrathoracic hæmorrhage, and possible damage to essential structures. There is usually severe shock, which should be treated as described in the first section.

Closed-circuit endotracheal anæsthesia with controlled breathing is essential, as it is important to maintain

adequate oxygenation with minimal lung movement to facilitate the work of the surgeon. Usually the pleura has to be opened, and reexpansion of the lung at the end of the operation requires an air-tight circuit. In cases in which there is a massive hæmorrhage from one lung, as in tuberculosis or hydatid disease, one-lung anæsthesia may be necessary.

The anæsthetic agent used should be cyclopropane given by the endotracheal or endobronchial route after a "Pentothal" and curare induction. During anæsthesia it is important that intermittent aspiration of the trachea or bronchus be performed. At the conclusion of the operation bronchoscopy is usually indicated.

If pneumonectomy or lobectomy is performed there is a sudden decrease in the volume of the pulmonary capillary bed; and the rate of blood transfusion should be decreased accordingly when the pulmonary vessels are clamped.

Post-operatively oxygen should be administered as described above, and coughing and breathing exercises instituted.

Head Injuries.

Head injuries present special problems because of the difficulty of assessment of the patient's condition, and the effects of cerebral damage and raised intracranial pressure. Shock and hæmorrhage should be treated as described earlier, except that intravenous therapy is used sparingly in the presence of high intracranial tension. In cases of ruptured cerebral aneurysm, pre-operative exsanguination to decrease hæmorrhage has given good results. After operation blood must be replaced until there is a hæmoglobin concentration of at least 90%. The operation is usually performed under local analgesia, which may be supplemented by continuous "Pentothal" administration or "Trilene" given by the endotracheal route if the patient is restless or shows signs of cerebral irritation. It has been stated (Minnitt and Gillies, 1948) that adrenaline in the local analgesic solution must not be used with "Trilene" because of the danger of ventricular fibrillation, but no difficulty from this cause has been encountered.

For facio-maxillary injuries requiring plastic repair, in the absence of gross intracranial injury, ether should be given by the endotracheal route with or without pharyngeal packing.

In cases of central block fractures of the maxilla, the exact extent of injury must be ascertained during the pre-operative consultation with the surgeon, so that no further damage occurs during intubation. This is performed under small doses of thiopentone and full doses of relaxing agents.

In fractures compound into the mouth—for example, fractured base of the skull—with aspiration of blood and vomitus, it is necessary to aspirate the trachea and nasopharynx before operation by means of an endotracheal tube and soft sterile rubber catheter.

Post-operative tracheotomy may be needed to permit intermittent aspiration of trachea and bronchi. Bronchoscopy is not advisable in cases of intracranial injury because of the increase in intracranial pressure consequent upon the necessary hyperextension of the neck.

Discussion.

From this detailed discussion, certain general principles emerge. Before operation an effort must be made to restore as far as possible the general condition of the patient. In some conditions—for example, intestinal obstruction with altered body fluid and electrolyte pattern from persistent vomiting—this may be done in part before operation; in others—for example, ruptured ectopic pregnancy—this is not possible until surgical repair of the causal lesion has been effected. During anæsthesia the prime consideration is adequate oxygenation. Under-ventilation is the most important cause of severe operative shock, which may easily become irreversible. If the patient is on controlled breathing, it is extremely important to ensure that under-ventilation does not take place. For this reason we have not employed assisted respiration. Adequate precautions must be taken against vomiting during induction of anæsthesia. Should this occur, treatment must be instituted immediately as outlined above.

So far no mention has been made of the use of local or regional analgesia in emergency surgery. It is of value mainly for limited procedures of short duration on moribund patients; but it has been used rarely in this series, because we consider that the mental and physical trauma involved is more detrimental to the patient than a suitably selected general anæsthetic. With local analgesia, exposure obtained by the surgeon is less, the duration of the operation is usually increased, and such anæsthesia is unsuitable when laparotomy is indicated without precise diagnosis. It is most unwise to use it if the surgeon is inexperienced in operating under conditions in which local analgesia is used.

The choice of anæsthetic is further restricted if diathermy is employed. In this case nitrous oxide, "Pentothal" or "Trilene" may be used. However, if the general condition of the patient contraindicates these anæsthetic agents because of the relatively low oxygen concentration that can be maintained, it is considered advisable to sacrifice the use of diathermy.

It should be emphasized that in all cases a full discussion with the surgeon must be held before operation is commenced. From this an indication of the duration of operation and type of operative procedure expected is obtained, and one learns whether gross blood loss is anticipated. These factors must modify the type of anæsthetic chosen. The final plan of treatment adopted is thus decided by the whole team involved.

Summary.

The conditions producing anæsthetic danger in emergency surgery are classified, and the management of each is discussed. Emphasis is laid upon adequate resuscitation both before and during operation, and upon the maintenance of full oxygenation during anæsthesia. There must be team work between all concerned in the management of every case.

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Reports of Cases.

A CASE OF SQUAMOUS EPITHELIOMA OF THE TONGUE IN A YOUNG WOMAN, AGED NINETEEN YEARS.

By RICHARD FLYNN AND H. HARRIS,
Sydney.

THE report of the following case was prompted not only by the rarity of a tongue carcinoma in a person of the patient's age, but even more by the fact that almost ten years after the diagnosis was made the patient is still in perfect health with no evidence of recurrence or metastasis.

H.V., aged nineteen years, was admitted to the Royal Prince Alfred Hospital on July 10, 1941, complaining of a lump on the left side of the tongue. This had first become noticeable about eight weeks previously as a small pimple on the under surface, but had grown rapidly, until at the end of four weeks it measured almost an inch in diameter. During the next four weeks growth had been considerably slower. The lesion was not painful unless irritated by food or pressure, and there was no evidence of discharge or bleeding. The patient did not smoke, and no relevant information could be elicited either from an investigation of her personal habits or from the family history.

Examination of the tongue revealed an ulcer about an inch in diameter on the under surface of the left side, some one and a half inches from the tip (Figure I). The edges were rolled, the floor was covered with a greyish slough and the base much indurated. At this stage no glands were palpable in the neck. The Wassermann test and the Kline test were carried out; both results were negative. Dark-ground examination of a smear revealed no spirochætes.

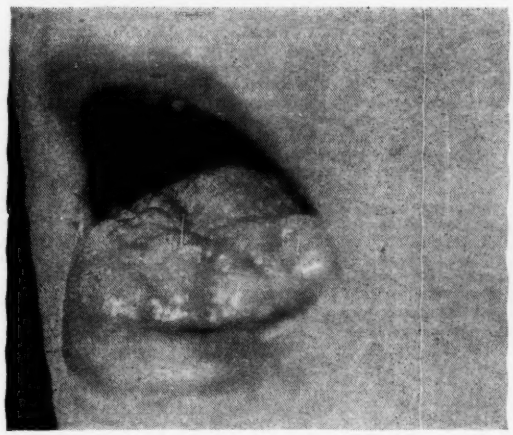

FIGURE I.
The ulcer.

On July 10 biopsy of the ulcerated tissue was performed and the pathologist's report was as follows. The specimen was a small, wedge-shaped piece of tissue from the under surface of the tongue. Microscopic examination of sections showed the presence of squamous epithelioma; the squamous cells were invading deeply into the tissues. Prickle cells could be plainly seen, and there was a moderate amount of keratinization. There was some small round-cell infiltration, very little fibrous tissue reaction.

A diagnosis of squamous epithelioma of the tongue was made.

On July 15 radium needles were inserted into the tongue so as to straddle the lesion—three needles containing four milligrammes, four containing two milligrammes and one containing one milligramme. The needles were removed on July 21 and the patient was discharged from hospital on July 22.

On September 2, 1941, the patient was readmitted to hospital with palpable glands in the neck, more noticeable on the left side. These were hard, but freely movable. On September 4 radical resection of the glands on the left side of the neck was carried out and the pathologist made the following report on specimens of excised tissue. On macroscopic examination the findings were as follows: (*a*) the lymph nodes were enlarged and appeared to be invaded; (*b*) the specimen consisted of a small amount of adipose tissue; no definite lymph node could be found. The microscopic findings were as follows: (*a*) the lymph nodes were seen to be invaded by squamous carcinoma; (*b*) the lymph nodes (two) were not invaded by squamous carcinoma.

Convalescence from this operation was uneventful, and on September 20 a similar radical resection was carried out on the right side. The pathologist's report on this occasion was as follows. (*a*) The large specimen consisted of a mass of adipose tissue, voluntary muscle and salivary gland; lymph nodes were also present, one of them being much enlarged. (*b*) The small specimen (supraclavicular gland) was bisected, and both halves were imbedded in one block. Microscopic examination gave the following results: (*a*) eight sections were examined, and in none was invasion by carcinoma detected; (*b*) in the lymph node no invasion by carcinoma was detected.

Convalescence was again without complication, and on October 23 deep X-ray treatment to the neck was commenced. A dose of 700*r* was administered to each side of the neck, and on October 28 the patient was discharged from hospital, with instructions to report to the follow-up clinic at intervals of six months.

FIGURE II.
The condition of the patient after operation.

On each occasion that the patient presented herself to the clinic, a thorough examination, including X-ray films of the chest, was carried out, and at no time was any evidence of recurrence or metastasis found. The most recent examination was performed in August, 1950, some nine years after the original diagnosis was made. The patient is still in perfect health, and there is no evidence whatever of the disease. In view of the length of time since operation, and of the thoroughness of subsequent investigation, it must be assumed that the condition has been cured (Figure II).

Reviews.

THE MEDICAL TREATMENT OF GASTRO-INTESTINAL DISORDERS.

"Medical Management of Gastrointestinal Disorders", by Garnett Cheney, M.D., clinical professor of medicine, Stanford University, is a book written primarily for medical students.[1] The author attacks the problem from the aspect of symptomatological diagnosis and treatment, and therefore of necessity covers a lot of ground twice. There is nothing new or startling in his description, but it is good straightforward material suitable for student teaching.

Some suggested methods of investigation, such as a daily enema for observing the colour of the stools in hæmatemesis, and light pounding of the abdomen with the clenched fist in the diagnosis of hepatitis, seem a little drastic. Nor do we agree that hæmatemesis patients should be got out of bed on the seventh day, with the use of aspirin in gastritis, nor with the injection of one millilitre of adrenaline intravenously for severe angioneurotic œdema.

It is also stated that clouding of the mind, disorientation and coma in a jaundiced patient are indicative of irreparable hepatic damage; no mention is made of the encephalitis of catarrhal jaundice, which has not a bad prognosis.

One of his quoted cases also does not ring true—that of a man treated for pernicious anæmia for many years with liver, who was given a blood transfusion which was followed by jaundice. Subsequent to his recovery, he had a complete remission of his pernicious anæmia.

It is these minor things which mar a book otherwise to be recommended for students.

SURGERY FOR NURSES.

A welcome addition to the list of nursing text-books is "Surgery for Nurses" by James Maroney.[2] This work comprises some 644 pages and covers all branches of surgical nursing, so that nurses can use it for their studies, whether they are working in a general surgical ward or in wards devoted to the various surgical specialties, such as thoracic surgery or orthopædics. Chapters are also devoted to such topics as pre-operative and post-operative care of the patient, sterilization and theatre technique, bacteriology and shock. In fact the scope of the work is most comprehensive. As happens with many of the books published by the house of Livingstone, the illustrations are very numerous, there being no fewer than 563; and as these are all carefully chosen to illuminate the text, their value to the student is great. In an endeavour to be complete, the author has included a chapter headed "Introduction to Obstetrics", but it is far from complete; it is to be hoped that in future editions this will be either greatly enlarged or else omitted. Apart from this chapter, the book is very well balanced, there being just enough theory to give the necessary background for the practical application of the principles of practical nursing; it is the practical outlook of the book that will make it a most valuable aid to all nurses.

ARTICULAR AND SPINAL DISEASES.

D. H. Collins, reader in clinical pathology in the University of Leeds, has written a book on the pathology of articular and spinal diseases which fulfils its intended aim of presenting an up-to-date and adequately illustrated account of the morbid anatomy of the joints and spine.[3] After a brief introductory consideration of the normal anatomy and histology, written from a refreshingly functional viewpoint, there follows a short review of those bone diseases which are particularly associated with lesions of the joints or spine. It is to the consideration of those that the book is limited. This section could well be expanded. A discussion of trauma in relation to joint disease is then succeeded by chapters in which the major articular diseases are separately considered. A special chapter is allotted to an up-to-date discussion of rheumatoid arthritis from the ætiological viewpoint. The section of the book devoted to the spine includes a separate and detailed account of the effects of disease of the intervertebral disks. Dr. Collins writes in a clear and attractive style. There are almost two hundred well reproduced illustrations, all original. Many of them, particularly the photomicrographs, are of high quality. A short list of references is given at the end of each chapter. This book should prove of great value to both undergraduates and practitioners who wish to know something of a subject which has often in the past been inadequately or obscurely treated.

[1] "Medical Management of Gastrointestinal Disorders", by Garnett Cheney, M.D.; 1950. Chicago: The Year Book Publishers. 8" x 5¼", pp. 484, with 29 illustrations. Price: $6.75.

[2] "Surgery for Nurses", by James Maroney, M.B., Ch.B., F.R.C.S. (England), L.R.C.P. (London), with a foreword by Miss Dorothy M. Smith, O.B.E.; 1950. Edinburgh: E. and S. Livingstone, Limited. 8½" x 5½", pp. 650, with 563 illustrations. Price: 27s. 6d.

[3] "The Pathology of Articular and Spinal Diseases", by Douglas H. Collins, O.B.E., M.D. (Liverpool); 1949. London: Edward Arnold and Company. 9" x 6", pp. 344, with 199 illustrations. Price: 35s.

EMERGENCY SURGERY.

Part III of Hamilton Bailey's "Emergency Surgery" reaches the usual high standard of this now classic publication.[1] This section deals with the emergency surgery of the rectum and anus, the male and female genito-urinary systems, and some post-operative complications. A chapter on surgical emergencies in obstetric practice by F. R. W. K. Allen has been introduced. Here it is surprising to find a description of the operation of symphsiotomy. Surely there is no place for this in modern obstetrics. It should be mentioned only to be condemned. A valuable chapter on abdominal emergencies in the tropics has been written by Professor A. T. Andreasen. *Phlebothrombosis decubiti* has now achieved the dignity of a chapter to itself. Here the use of the anticoagulants and venous ligation is well described. It is puzzling, however, to find no reference to the important work of Cummine and Lyons, of Sydney, on this subject. There is no mention, for instance, of the fact that the majority of lung thromboses may be of primary origin in the vessels at the lung bases and not embolic at all. There is little else to criticize apart from the presence of some unnecessary illustrations of commonly used surgical instruments and everyday manœuvres.

APPLIED MEDICINE.

"Applied Medicine", by G. E. Beaumont, of the Middlesex and Brompton Hospitals, represents a new technique in didactic medical writing and its style is so delightful that it is difficult to imagine that it came from the same pen as that of the author of the well-known medical text-book.[2] It springs from the rich experience of twenty-five years of private and hospital practice and deals with problems in diagnosis and treatment of individual cases involving the application of theoretical medical knowledge together with the use of laboratory tests and investigations. The book is divided into four parts. Part 1 consists of descriptive cases covering 33 medical conditions of such wide variety as disseminated *lupus erythematosus*, persistent spontaneous pneumothorax, masked thyreotoxicosis and aortic aneurysm in a woman. The histories really live and some in fact are quite dramatic; the physical examinations are given in adequate detail, the results of relevant investigations are recorded and the author's comments are in most cases highly instructive. Part 2 consists of short notes on cases and is really a continuation in an abridged form of Part 1. Each case is chosen for the lesson which it teaches, and is presented in a way which impresses it on one's memory. Part 3 consists of cases demonstrated at the bedside by question and answer and is a selected record of teaching rounds. It forms an excellent model for clinical teachers, though at times they are inclined to be a little tedious and make one realize that the English undergraduate is much the same in wisdom and knowledge as his Australian

[1] "Emergency Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., assisted by Norman M. Matheson, M.B., Ch.B., M.R.C.P. (London), F.R.C.S. (England), F.A.C.S.; Part III; Sixth Edition; 1950. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 10" x 6½", pp. 167, with 246 illustrations, some of them coloured. Price: 21s. per part.

[2] "Applied Medicine", by G. E. Beaumont, M.A., D.M., F.R.C.P., D.P.H.; 1950. London: J. and A. Churchill, Limited. 9" x 6¾", pp. 556, with 74 illustrations. Price: 30s.

brother. Part 4 is a short section entitled "Idle Thoughts". It is somewhat reminiscent of Gee's "Aphorisms" and contains a considerable amount of potted wisdom.

The book is well illustrated with radiographs, relevant clinical photographs, diagrams and charts, and its nature is such that it can be picked up and read with profit at almost any point. It would form an excellent companion to any medical text-book and should prove both stimulating and instructive to undergraduate and post-graduate students alike.

MEDICINE AND MANKIND.

SINCE the appearance of its first edition in 1941, "Medicine and Mankind", by Arnold Sorsby, has become well known. A second edition (brought up to date) has now appeared in quite handsome form.[1] The author's object is to review for the non-medical reader the activities of medicine in the light of basic modern conceptions of health and disease. The essential interrelationship of man with his internal, his social and his natural environments is stressed. The generalization and simplification inevitable in a book of this type lay it open to criticism. It would be quite impossible for the author to avoid statements and views not universally acceptable. However, he expresses the position as he sees it and the result is readable and thought-provoking. The book will be appreciated by any intelligent layman with some interest in the subject; most medical practitioners will find a good deal of the material too elementary, though the two concluding chapters contain many facts, figures and thoughts on the social aspects of health and disease that make them worth reading.

A TEXT-BOOK OF OBSTETRICS.

WARM CONGRATULATIONS are due to Professor Bruce Mayes on the excellence of his new publication "A Textbook of Obstetrics", in which he presents the story of modern obstetrics in a new and pleasing fashion.[2] The inclusion of contributions by other well-known Australians and the abundant references to Australian research give to the book a local colour which should appeal at once to students and practitioners in this country.

The traditional approach according to the powers, the passages and the passenger has not been followed, the subject being presented in a highly practical yet simple way. Special conditions are dealt with by colleagues who are qualified authorities, such as "Thyrotoxicosis in Pregnancy" (Poate), "Leucorrhœa in Pregnancy" (McIlrath), "Radiology in Obstetrics" (Maitland) and "Intravascular Thrombosis" (Cummine).

In spite of its size (1040 pages) the content of the book is well within the ambit of the general practitioner and, therefore, eminently suitable for the student. It is lavishly illustrated and in all respects up to date. Thus we find full descriptions of the Aschheim-Zondek, Friedman and Hogben pregnancy tests under "Symptoms and Signs of Pregnancy", the biphasic temperature chart of the menstrual cycle and pregnancy under "Maternal Changes due to Pregnancy", the effect of heparin on the prevention of red cell thrombi in eclampsia, and reference to the recent theory of Roland Beard on the occipito-posterior position.

The author deals with the abnormal presentations in clear-cut though didactic fashion and rightly discounts traditional techniques when experience has proved them worthless. Hence the conversion of a brow to face or occiput presentation is rejected in favour of internal version or Cæsarean section, since experience shows that brow presentation is usually diagnosed late in labour when the apparently simpler conversion is impossible. Breech delivery is impressively detailed, and the author's enthusiasm for the Lovset manœuvre is manifest, ten beautiful illustrations ensuring a perfect understanding of the method. Delivery of the head is by the Mauriceau-Smellie-Veit procedure, no mention being made of the Prague seizure, except for occipito-posterior positions.

Discussion of the toxæmias of pregnancy occupies four chapters in which modern concepts of causation and treatment are expounded. Stress is laid upon the fallacy of a reduced protein diet in preeclamptic toxæmia, a régime which still lingers even with some who should know better. Some will disagree, however, with the dictum that the eclamptic should be kept in bed till the urine is free of albumin and the blood pressure approaches normal—this might be for ever. It is surprising also that the author questions the existence of fulminating eclampsia, a rare but undoubted entity which from time to time keeps us humble.

The chapters on pelvic morphology and measurement and their relationship to labour are enjoyable and conclude with a contribution by Cain on "The Australian Female Pelvis".

Antepartum hæmorrhage, its causes, effects and treatment, are extremely well presented. The external and concealed types of accidental bleeding are, in practice, intimately blended—so also in the discussion under review. This is further evidence of the practical mind of the author.

In the treatment of *asphyxia neonatorum* the Drinker apparatus is elaborately described, and its superiority over older methods of resuscitation emphasized.

The Rhesus factor is dealt with very simply, as also are the various techniques of Cæsarean section, this being in accord with the author's aim to produce a book for student and practitioner, which, in fact, he has successfully achieved. For advanced techniques omitted from this volume the author refers the reader to his former publication "Practical Obstetrics".

Notes on Books, Current Journals and New Appliances.

THE ROYAL MELBOURNE HOSPITAL MEDICAL MANUAL.

THE first edition of "The Royal Melbourne Hospital Medical Manual", well printed and bound with an attractive cover, should prove acceptable and useful.[1] It includes the hospital pharmacopœia, and sections on clinical pathology, clinical biochemistry, dietetics and radiology. The sections on clinical pathology and biochemistry are primarily concerned with a description of the preparation of patients for certain tests, the collection of specimens in the wards and out-patient departments, and the interpretation of the tests when completed. The notes on radiology relate to the preparation of patients for radiological examination. The section on dietetics seeks to incorporate the latest knowledge on the subject. Although a few points in the manual apply specifically to the Royal Melbourne Hospital, most of the information is of general interest and application. The editor has produced a convenient and informative handbook.

MODERN TREATMENT.

THE general practitioner seeking practical information on modern treatment, presented briefly enough for the man with little time for reading and yet in enough detail to be of clinical use, will find the "Modern Treatment Year Book, 1950" acceptable.[2] The editor, Sir Cecil Wakeley, has assembled 38 chapters, each on a subject of interest and by a practitioner of good standing. Appropriate information on ætiology, pathology and diagnosis is included in some chapters, but this is not allowed to lengthen the chapters unduly or to interfere with the main purpose of the book. The treatment is described of bronchiectasis, heart abnormalities (from the surgical aspect), schizophrenia, sinusitis, keratitis and corneal ulcers, prostatic enlargement, angina of effort, enuresis, influenza, brachial neuritis, iridocyclitis, obesity, subacute bacterial endocarditis, corneal ulcers, early

[1] "Medicine and Mankind", by Arnold Sorsby; Second Edition; 1950. London, New York, Toronto: Staples Press, Limited. 8″ × 5¼″, pp. 232, with 36 illustrations. Price: 12s. 6d.

[2] "A Textbook of Obstetrics", by Bruce T. Mayes, M.V.O., M.B., B.S. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S., F.R.C.O.G.; 1950. Sydney, Wellington and London: Australasian Publishing Company. 9″ × 6¾″, pp. 1062, with 537 illustrations, some of them coloured. Price: £5 10s.

[1] "The Royal Melbourne Hospital: Medical Manual", edited by Ivan Maxwell, M.D., B.S., M.Sc., B.Agr.Sc., F.A.C.I., F.R.A.C.P.; 1949. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 6¼″ × 4¼″, pp. 304. Price: 20s.

[2] "Modern Treatment Year Book, 1950: A Year Book of Diagnosis and Treatment for the General Practitioner", edited by Sir Cecil Wakeley, K.B.E., C.B., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S.; 1950. London: Baillière, Tindall and Cox. 8¼″ × 5¼″, pp. 388, with 23 illustrations. Price: 17s. 6d.

ankylosing spondylitis, chronic valvular heart disease, *alopecia areata*, depression, deafness, torticollis, acute rheumatism in childhood, tuberculosis of the hip joint in children, alcoholism, venereal diseases, anæmia, renal calculus, epiphora, common complications of labour, menstrual disorders, sinusitis associated with allergy, sciatica, heart disease, osteoarthritis, rheumatic diseases, and pulmonary tuberculosis; separate chapters deal with the indications for gastrectomy, trends in fracture surgery, and spa treatment. It is a pity that the individual chapters are not grouped in any way (the order we have listed is practically that of the book), but there may be good editorial reasons for this; to obtain and assemble 38 articles, each from a separate author, and to arrange their publication under one cover quickly enough to maintain the current value demanded of an annual volume, is no mean task.

ADVANCES IN SURGERY.

THE second volume of "Advances in Surgery" contains only 11 articles, and as the volume has nearly 600 pages, the articles cover their subjects fairly comprehensively.[1] Indeed, as is stated in the preface, certain articles are really "monographs on subjects in which a great deal of experience has accumulated over a long period in a laboratory of surgical pathology or under the extraordinary concentration of cases during the war". Many well-known names are included among the authors. R. L. Riley and A. F. Cournand write on pulmonary function studies in relation to chest surgery, R. H. Sweet writes on surgery of the œsophagus, R. H. Smithwick on surgical treatment of hypertension, A. O. Whipple on portal bed block and portal hypertension, G. H. Humphreys on surgery of congenital anomalies of the heart and great vessels, A. E. Walker and R. G. Fisher on the treatment of cranio-cerebral injuries, E. J. Pulaski on streptomycin in surgical infections, Bradford Cannon and J. B. Brown on skin grafting, N. C. Foot on the pathology of peripheral nerve tumours, L. G. Lewis on tumours of the testis, and C. G. Child on the management of pancreatico-duodenal ulcers. The articles are well illustrated and documented. Surgeons interested in the subjects listed should find the book of much interest and value.

"GROWING GIRLS."

"GROWING GIRLS" is a sensible little film dealing with female adolescence and more particularly with the onset of menstruation. In a natural way the changes of adolescence are explained as the growing-up process preparatory to potential motherhood. The basic facts of menstruation are shown with the aid of diagrams, and simple rules of hygiene are detailed. It is all quite matter-of-fact and informative, but at the same time is human in its approach and does not offer more information than is required for its purpose. The people—women and girls—who appear in it are all healthy-looking folk, and the normality of menstruation is assumed rather than stressed. Minor faults might be found, but they do not affect the practical value of the film, which can be commended as suitable for showing to girls in their early teens. The photography and sound are good. The running time is seven to eight minutes. The film is available for hire or for purchase from the Father and Son Welfare Movement, 242 Pitt Street, Sydney.

ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION, 1949.

THE proceedings of the annual meeting of the British Medical Association for 1949 have now been published.[2] The volume includes the presidential address, "Man and the Machine", by C. W. Curtis Bain (printed in full), a report of the popular lecture, "Health and Happiness", by Charles Hill, and at least one complete paper on each subject considered in the various sections; in addition, summaries are presented of other papers read at the meeting, with an account of the discussion in some cases. A wide range of subjects was covered, so that the extensive index is useful and necessary. The volume, well printed and bound, will be a permanent record of value.

[1] "Advances in Surgery", edited by William DeWitt Andrus *et alii*; Volume II; 1949. New York: Interscience Publishers, Incorporated. London: Interscience Publishers, Limited. 9″ × 6″, pp. 600, with many illustrations. Price: $11.00.

[2] "Proceedings of the Annual Meeting, 1949: British Medical Association"; 1950. London and Sydney: Butterworth and Company (Publishers), Limited. 9¼″ × 6½″, pp. 482, with 56 illustrations. Price: 24s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Rhesus Danger: Its Medical, Moral and Legal Aspects", by R. N. C. McCurdy, M.B., Ch.B., D.P.H.; 1950. London: William Heinemann (Medical Books), Limited. 7¼″ × 5″, pp. 146. Price: 5s.

Intended for medical and lay readers.

"The Ethical Basis of Medical Practice", by Willard L. Sperry, with a foreword by J. Howard Means, M.D.; 1950. London and New York: Paul B. Hoeber, Incorporated. 8″ × 5½″, pp. 186. Price: $2.50.

The author is Dean of the Harvard Divinity School and the book was evolved from a lecture to house officers at the Massachusetts General Hospital.

"Medicine and Mankind", by Arnold Sorsby; Second Edition; 1950. London, New York, Toronto: Staples Press, Limited. 8″ × 5¼″, pp. 232, with 36 illustrations. Price: 12s. 6d.

A survey of medicine for the medical student and the general reader.

"A Study of Diphtheria in Two Areas of Great Britain, with Special Reference to the Antitoxin Concentration of the Serum of Inoculated and Non-Inoculated Patients and Other Persons; and the Relation of This to the Incidence, Type and Severity of the Disease"; Privy Council, Medical Research Council Special Report Series Number 272; by Percival Hartley, W. J. Tulloch, M. Anderson, W. A. Davidson, J. Grant, W. M. Jamieson, C. Neubauer, R. Norton and G. H. Robertson; 1950. London: His Majesty's Stationery Office. 9½″ × 6″, pp. 166. Price: 4s. net.

An attempt to discover why immunization does not always confer complete protection against diphtheria.

"The 1949 Year Book of Endocrinology, Metabolism and Nutrition (December, 1948-January, 1950)"; Endocrinology edited by Willard O. Thompson, M.D.; Metabolism and Nutrition edited by Tom D. Spies, M.D.; 1950. Chicago: Year Book Publishers, Incorporated. 7″ × 4¾″, pp. 550, with illustrations. Price: $4.75, post paid.

One of the Practical Medicine Series of Year Books.

"Annual Review of Medicine", by Windsor C. Cutting and Henry W. Newman; Volume I; 1950. Stanford: Annual Reviews, Inc. 9″ × 6″, pp. 452. Price: $6.00.

The scope of the articles is intended to be broad and the authors include their own critical assessment.

"Physician's Handbook", by Marcus A. Krupp, M.D., Norman J. Sweet, M.D., Ernest Jawetz, Ph.D., M.D., and Charles D. Armstrong, M.D.; Sixth Edition; 1950. California: University Medical Publishers. 7″ × 4″, pp. 388, with illustrations. Price: $2.50.

A pocket reference book for student and physician.

"A Short Textbook of Surgery", by C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S. (Edinburgh); Fifth Edition; 1950. London: J. and A. Churchill, Limited. 9¼″ × 6¼″, pp. 684, with many illustrations. Price: 30s.

A completely revised edition with new sections on the surgery of pulmonary stenosis and portal hypertension.

"Researches on the Radiotherapy of Oral Cancer", by Constance A. P. Wood and J. W. Boag (with P. Howard-Flanders, A. Glücksmann, L. H. Gray and F. G. Spear); Medical Research Council of the Privy Council, Special Report Series Number 267. London: His Majesty's Stationery Office. 9½″ × 6″, pp. 148, with illustrations, some of them coloured. Price: 12s. 6d.

A report of investigations from the radiological and histological aspects with a statistical analysis and a discussion of future research in radiotherapy.

"Adrenal Cortex: Transactions of the First Conference, November 21-22, 1949", edited by Elaine P. Ralli; Associate Editors, Konrad Bloch and Gregory Pincus; 1950. New York: Josiah Macy, Junior, Foundation. 9″ × 6″, pp. 190, with illustrations. Price: $2.00.

Five papers on the adrenal cortex and associated hormones with the discussion that followed their presentation.

The Medical Journal of Australia

SATURDAY, OCTOBER 7, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE TREATMENT OF PULMONARY HYDATID DISEASE.

THE study of hydatid disease has led to some remarkable controversies. For example, for many years the nature and ætiology of the infiltrating alveolar type of hydatid cysts in the liver was a very vexed question: some, the "dualists", held that it was due to a distinct species of tapeworm; others, the "monists", that it was due to *Tænia echinococcus*, manifesting itself in a certain atypical way. The leading authorities on the subject, including F. Dévé and H. R. Dew, assert that the question has now been resolved in favour of the monists, but there are one or two eminent students of the subject still unpersuaded. The most recent controversy, one which should be followed with close attention in this country, concerns the surgical treatment of pulmonary hydatid disease. A body of opinion appears to be growing in England and America in favour of pulmonary resection for hydatid cysts of the lung. The modern views on the subject were crystallized several years ago by N. R. Barrett, of London, in a paper based on the treatment of 30 patients: he discussed the pros and cons of conservative treatment and set out certain fairly comprehensive indications for lobectomy.[1] L. A. Brewer III, W. M. G. Jones and F. S. Dolley, of Los Angeles, in an article on non-malignant intrathoracic lesions,[2] also based on a consideration of 30 cases, only one of which, however, proved to be a hydatid cyst, express a much more radical view. "Since the best treatment for echinococcus disease is pulmonary resection", they state, "if the condition of the patient permits, pulmonary resection is the treatment of choice". "With this advice I heartily disagree", writes M. P. Susman, of Sydney, who has taken up the cudgels in favour of more conservative surgery, and who writes in a tone of much assurance from personal experience of 39 cases and a consideration of 45 cases culled from hospital records.[3,4]

[1] *Thorax*, March, 1947.
[2] *The Journal of Thoracic Surgery*, August, 1948.
[3] *Thorax*, June, 1948.
[4] *The Journal of Thoracic Surgery*, March, 1950.

Susman points out that hydatid cysts may be coughed up uneventfully, but that various dangers attend intrabronchial rupture, such as anaphylaxis, asphyxia, hæmorrhage and suppuration. Hence he believes that all cysts situated peripherally in the lung and all hilar cysts, unless small, should be treated promptly by operation. These last should also be treated surgically if they are not coughed up and if they grow to two inches in diameter. He gives the aims of conservative surgery as "to remove the cyst completely so that only the adventitia remains, to avoid implantation of hooklets and scolices and to prevent complications such as infection of the pleural cavity and lung, tension pneumothorax, atelectasis and delayed re-expansion of the lung". He favours a one-stage operation and summarizes as follows the treatment which he recommends.

When the lung is adherent to the chest wall:

(*a*) If the cyst is not infected—
The contents are removed and the adventitia is closed.

(*b*) If the cyst is infected—
The contents are removed and the cyst space is drained. Should the pleural cavity be entered during the operation it must be securely closed before opening the adherent cyst.

When the lung is not adherent:

(*a*) If the cyst is not infected—
The pleural cavity is freely opened and protected, the contents of the cyst are removed, the adventitia is closed and sutured to the chest wall and the pleural cavity is drained, with a water seal, through a stab wound.

(*b*) If the cyst is infected—
The pleural cavity is opened and protected, the contents of the cyst are removed, the cyst space is drained and marsupialized and the pleural cavity is drained through a stab wound.

Susman states that lobectomy may be necessary for certain conditions which he enumerates under five headings. The first of these is the occurrence of serious hæmorrhage from the cyst space during the course of a conservative operation. The second condition is residual bronchiectasis if the symptoms are so severe as to warrant lobectomy. The third condition is an empty sac in the presence of severe hæmorrhage or infection. The fourth condition exists when the diagnosis is uncertain and the fifth when the cyst is a giant cyst. In discussing Barrett's indications for lobectomy Susman states that no operation at all is necessary for dead cysts or for empty sacs uncomplicated by hæmorrhage or sepsis, while complete removal of the endocyst and adequate drainage are all that is necessary for infected cysts. The drainage must be adequate or there may result the persistent sinuses which Barrett cites as one of the reasons for resort to lobectomy in the first instance. The mortality from the conservative operation is very small, no deaths from sepsis having occurred in the cases under review. It would appear that the possibility of residual bronchiectasis may have influenced American surgeons in their predilection for lobectomy, for an editorial footnote to Susman's paper in *The Journal of Thoracic Surgery* indicates that the author was questioned about his failure to present post-operative bronchograms. The reply was that these were not made as a routine because all but two of the patients recovered quickly and apparently completely; in the two exceptions bronchograms were made and found to be normal. Another complication, and sometimes an alarming one, which may be avoided by lobectomy, is the

serious hæmorrhage which may follow the opening of any hydatid cyst. It is possible that in the future local application of thrombin will make this complication less formidable.

With the existing unsettled state of surgical opinion about the treatment of pulmonary hydatid cysts, a recent review by N. Waddle, of Dunedin, of 478 cases reported in the Louis Barnett Hydatid Registry of the Royal Australasian College of Surgeons, is very valuable and welcome.[1] This review is comprehensive and succinct, dealing with incidence, symptomatology, diagnosis, surgical pathology, management and treatment. Ninety-six of the 478 patients were traced and examined in a "follow-up" survey, conducted for the most part a good many years after the patients had originally come under treatment. It is stated that the operative mortality rate for 362 patients treated surgically was 6%, which seems very low in view of the fact that the results are those of many different surgeons throughout Australia and New Zealand and that many of the operations were performed before the invention of the modern methods of anæsthesia. No attempt has been made to differentiate between the results of operations performed in the several decades covered by the review. Another finding was that the vast majority of ex-patients remained free of symptoms and physically fit in after years. Many of the patients followed up were examined by bronchography, and permanent dilatation of the bronchi could usually be demonstrated; but this bronchiectasis remained symptomless in the great majority of cases and was not apparent in plain X-ray films. Waddle is of the opinion that the bronchiectasis requires no treatment, unless it should become the seat of infection later. Waddle concludes also that "gross disorganization of the lung, resulting from infective processes which have complicated a ruptured hydatid cyst either before or after its removal, is the only indication for lobectomy or pneumonectomy". He finds no justification for these procedures in the treatment of hydatid disease save in such an exceptional circumstance as the occurrence of severe hæmorrhage otherwise uncontrollable during the removal of a cyst.

It is an interesting commentary on the great technical advances made in thoracic surgery in recent years that an operation so heroic as lobectomy should be preferred by some to a simpler and less risky one which can do as much for the patient. The explanation no doubt is that lobectomy, being radical, clean and more or less stereotyped, appeals to thoracic surgeons of experience who do not frequently encounter pulmonary hydatid disease and who are aware of the surgical pitfalls which the conservative operation presents. Perhaps the clear exposition here quoted, which is not yet to be found in text books, of what to do in the various possible circumstances, will yet tip the scale in favour of conservative surgery that will leave the patient in possession of his two lungs. Charles Lamb in his well-known "Dissertation" has related how the ancient Chinese would burn down a house to roast a pig.[2] This custom, he states, continued till in process of time a sage arose who made a discovery that the flesh of swine, or indeed of any other animal, might be cooked without the necessity of consuming a whole house to dress it.

[1] *The Australian and New Zealand Journal of Surgery*, May, 1950.

[2] *The London Magazine*, September, 1822.

Current Comment.

DIPHTHERIA RESEARCH IN GERMANY.

WORKERS in many parts of the British Commonwealth have wondered about biological research in Germany during and after the Great War, and have found little indication of activity. Now, however, comes a series of highly interesting and provocative papers from G. Wildfuhr, of the Institute of Hygiene, University of Leipzig, abstracted in some detail by J. W. McLeod in the *Bulletin of Hygiene*, Volume XXV, March, 1950. The subject is diphtheria, and the work was doubtless stimulated by the severe wartime epidemics in northern Europe and Scandinavia.

In six separate papers Wildfuhr submits evidence in support of the following facts.

1. He has recovered diphtheria bacilli from the blood in five out of 23 fatal cases of diphtheria by repeated culture of the blood in broth during the forty-eight hours before death.

2. He has followed 202 contacts of patients with diphtheria, and among 31 persons who actually contracted the disease he recovered diphtheria bacilli from the blood of 28. These cultures were obtained before throat manifestations made the clinical diagnosis obvious; the patients were all febrile, and local signs appeared within three days. Blood taken after this failed to yield the organism.

3. He records levels of diphtheria toxin circulating free in the blood of severely ill patients, both in the pre-paralytic and the early paralytic stages. In seven out of 16 in the first group and in three out of 37 in the second group toxin could be demonstrated. The highest titres recorded were 0·8 to 1·33 minimal lethal doses per 100 millilitres of serum, and these figures were found in patients with rapid ascending and progressive types of paralysis. The patients also showed free toxin in the cerebro-spinal fluid to the titre of 0·02 to 0·03 minimal lethal doses per 100 millilitres of fluid. Antitoxin titres in the serum were observed, and found to be lowest in individuals in whom free toxin had been demonstrated earlier, and highest in subjects in whom no paralysis developed. Studies of the toxicity and virulence of the strains of diphtheria bacilli isolated were carried out—55% of *gravis* and 30% of *mitis* strains fell in the high toxin producing group *in vitro*. By the use of an *in vivo* test on the rabbit cornea, toxin could be shown in the serum of a guinea-pig twelve hours after it had been inoculated with a virulent *gravis* strain; the time interval was much longer after a *mitis* strain had been used for the inoculation. Virulence was tested by subcutaneous inoculation of a small standard dose subcutaneously into the guinea-pig. Strains considered fully virulent killed the animals within four days; the others were graded accordingly. By this criterion 85% of the *gravis* and 30% of the *mitis* strains were fully virulent.

4. Passage experiments from animal to animal were carried out on a selected group of strains, all of low virulence; both normal and immunized guinea-pigs were used. While no change was recorded in the *gravis* strains and four *mitis* strains, the remaining two *mitis* strains are stated to have become highly virulent and eventually developed the characteristics of *gravis* strains in the fermentation of polysaccharides. The author considers that these experiments explain the many "atypical strains" encountered by workers endeavouring to type *Corynebacterium diphtheriæ* by means of colony form and fermentation of starch and glycogen; and he describes the change in the development of variants occurring in an immunized population.

Careful consideration should be given to some of these ideas. A terminal bacteriæmia has been postulated and sought many times before, perhaps not with the same persistence, and certainly not with the same success. The finding of diphtheria bacilli in the blood of no less than 28 of 31 persons who later developed the disease is an even more dramatic observation, and perhaps may find support in view of Fenner's observations on the primary

viræmia of ectromelia, demonstrated in the mouse. The presence of free toxin in the circulating blood and in the cerebro-spinal fluid of individuals becoming seriously paralysed conflicts with current views on the strong affinity of toxin for susceptible tissues, and the author suggests that toxin fixed by antitoxin administered early may be released, or that more may be formed in subjects whose active immunity has not adequately replaced the therapeutic antitoxin. The observation certainly provides a possible reason for the occasional benefit seen from a late administration of antitoxin in pædiatric practice. There is no mention of tests of antitoxin in the cerebro-spinal fluid, a point of further interest in this context.

The last paper is one which will arouse the greatest controversy amongst laboratory workers—the conversion of *mitis* to fully virulent *gravis* strains. There are no details of the difficult animal passage without intervening culture, and consequently no check upon the possibility of chance infection of the animals with the *gravis* strain and its replacement of the original *mitis*. We have abundant evidence of change of type in the pneumococcus, but then the transforming substance is a known chemical substance and can be controlled; the change is not effected by blind animal passage. There is no known qualitative difference in diphtheria toxin from different strains of bacilli, and the explanation of epidemic spread in the highly immunized Scandinavian countries is extremely puzzling, and these experiments do not throw any light on it.

Confirmatory evidence of this work may be difficult to obtain unless further severe epidemics occur, and confirmation is certainly needed before the evidence can be wholly accepted.

DANGEROUS UNIVERSAL DONORS.

IN the last few years more reports have been published of hæmolytic reactions resulting from the transfusion of group O plasma into recipients of group A. D. M. Ervin, R. M. Christian and L. E. Young, in making further observations on this subject, point out that reactions of this type are not so severe as those following the transfusion of whole blood of an incompatible group, and therefore not so easily detected clinically.[1] These authors previously reported the case of a group A recipient who suffered a severe and prolonged hæmolytic reaction due to group O blood containing α antibodies of the immune type. The purpose of their present paper is to call attention to a series of three patients who had definite hæmolytic reactions after administration of group O plasma or blood. The first of this series was not strictly speaking a patient, but a volunteer who received 250 millilitres of group O plasma on two occasions. The other two received whole blood, and had more intense reactions. Hæmaglobinuria was the most striking symptom in the latter, and further investigation showed the presence of spherocytosis, increase in osmotic fragility, and also mechanical fragility, and, what is even more significant, anæmia. The authors had further taken the precaution in one of their patients treated with whole blood transfusion of adding A and B substances to the O group blood so as to neutralize possible hæmolysins, but this was not effective, even though followed up by intravenous administration of another dose of the soluble substances after the passing of black urine had occurred. The further description of work carried out on sera containing immune anti-A and anti-B antibodies is rather outside the scope of this comment, which concerns more the clinical possibilities of incompatibility of this kind. Ervin, Christian and Young remark that, although the hæmolysin titre of immune anti-A antibodies is more readily reduced by soluble A factor than is the agglutinin titre, the addition of this factor to O group blood, or the intravenous administration of the factor may not make the use of the blood safe. They think that these precautions probably lessened the severity of the hæmolytic reaction, and suggest that perhaps the use of more concentrated preparations may be found effective. From the practical aspect the authors bring up important points. One is the advisability of discontinuance of the term "universal donor", though the difficulty inherent in a subclassification such as "dangerous universal donor" is evident, quite apart from the contradiction in terms. Another important point is that a hæmolytic reaction may occur without any of the familiar signs of incompatibility, such as chills, fever, pain in the back or flank, oliguria, or circulatory collapse. Even hæmoglobinuria and jaundice may not be seen. The further question of the practicability of screening "universal" donors is also discussed in this article. One expected difficulty is how to decide what importance should be attached to the presence of small amounts of these antibodies in O group plasma or serum. For the present it is wise to remember that there are exceptions to the "universal" acceptance of O group blood.

[1] *Blood*, June, 1950.

TWENTY-FIVE YEARS OF MEDICAL RESEARCH.

THE Thomas Baker, Alice Baker and Eleanor Shaw Medical Research Institute, Melbourne, was founded in 1926, and with the twenty-third annual report of the institute (covering the activities of 1949) the trustees have included a retrospect of the quarter-century. At the same time has been issued the first annual report of the Alfred Hospital Clinical Research Unit, which works in close collaboration with the institute and has with it a common director, T. E. Lowe. The institute developed directly from the laboratory services of the Alfred Hospital and was established to provide an efficient routine hospital laboratory service and facilities for medical research. The laboratories covered the fields of pathology, bacteriology, biochemistry and electrocardiography, but gradually as a matter of deliberate policy these services were handed over to the hospital staff as well-developed units. As is pointed out in the report, this aspect of the institute's activities illustrates an important phase of medical research—namely, the development of methods of investigating disease and their application to the management of individual patients; when this has been done, use of the methods becomes a matter of routine and should be left to the medical staff requiring such services. It is very satisfactory that the institute has been able not only to do this worthwhile service for the hospital, but also to be relieved of the burden of routine work in due time. This has enhanced the opportunity to carry on basic research into medical problems. Two of many successful pieces of research work carried out by the institute staff have been the researches into the biochemistry of *diabetes mellitus* and the development of the anticoagulant drug E.D.C. (reported by Fantl and Nance in this journal on August 2, 1947). Another useful piece of service has been the development of a number of new techniques for the hospital biochemistry service. The institute has sponsored the publication of three monographs that have become standard texts. It launched a campaign in 1929 aimed at the reduction of the mortality from *diabetes mellitus* in Victoria. During the recent war members of the staff were actively engaged in work with the Chemical Defence Board of the Munitions Supply Laboratory, and valuable contributions were made in, for example, the production of essential drugs. At the present time the basic research work in the institute is following two main lines: an investigation of the chemistry of blood coagulation and studies of the relationship of ductless glands and carbohydrate metabolism. In addition facilities and assistance are made available to honorary medical officers of the Alfred Hospital in various problems that arise, and the young clinical research unit is being aided in every way; the core of the long-term clinical research plan is the development of a cardio-vascular research centre. The institute's plans for the future should produce results at least equal to the creditable achievements of the past.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Quantitative Studies in Diphtheria Prophylaxis.

L. B. HOLT (*The British Journal of Experimental Pathology*, August, 1949) has studied the primary response as the beginning of quantitative studies in diphtheria prophylaxis. He states that the introduction of purified toxoid adsorbed onto aluminium phosphate has made possible accurate comparable reproducible studies in this field, and that experiments support the contention that a single primary inoculation of toxoid stimulates globulin-producing cells, which subsequently produce altered globulin-antitoxin, for a considerable period of time. The aluminium-adsorbed toxoid stimulus takes up to twelve days to reach a stabilized level, and during analysis of the experiments it was observed that increasing amounts of aluminium phosphate up to a maximum of three milligrammes increased the antitoxin response, without increase in the amount of toxoid it carried. Histological study of the nodule produced in the subcutaneous tissues by the inoculation shows that the injection site is slowly infiltrated with leucocytes and then becomes encapsulated, and the mineral carrier of the toxoid is present for a considerable period of time. Once the primary stimulus has acted for the period of ten days, surgical excision of the nodule does not alter the level of circulating antitoxin. It is suggested that the optimum transport of antigen to antibody-producing cells is attained in the first twelve days, and that those cells can genetically transmit to their daughter cells the ability to form antibody.

Feeding Tests with Enterococci.

G. M. DACK, C. F. NIVEN, J. B. KIRSNER AND HOMER MARSHALL (*The Journal of Infectious Diseases*, September-October, 1949) have studied the results on human volunteers of feeding tests with enterococci and tyramine. The investigation was concerned with cultures of enterococci used as "starters" in the manufacture of cheese, and the possibility that they might be agents of food-poisoning symptoms. Since it had been shown that enterococci can decarboxylate tyrosine with the production of tyramine, the effect of the administration of tyramine to the volunteers was also studied. Three strains of enterococci obtained from outbreaks of food poisoning were tested, as well as the strain of *Enterococcus fæcalis* used for the cheese "starter" material; they were administered in considerable amount to the volunteer subjects, all of whom were given a control test period of observation after a control feeding with either milk or broth. The results of 52 experiments on 37 volunteers showed that the administration of the cheese "starter" culture, and of tyramine in doses up to one gramme, was without effect on the experimental subjects. Two of the volunteers fed *Streptococcus fæcalis* cultures had definite symptoms of diarrhœa; one had in addition pain and nausea. The authors discuss the many other factors which might influence the development of food poisoning in the presence of a particular strain of organism.

Serological Studies in Rheumatic Fever.

EDWARD E. FISCHEL, RUTH H. PAULI AND JACK LESH (*The Journal of Clinical Investigation*, September, 1949) have continued their serological studies in rheumatic fever with an examination of serum complement in the rheumatic state. A series of estimations on normal five-year-old children was carried out in order to have a base line, and the figure obtained was 37·7 units (standard deviation 3·9 units), with a 50% hæmolysis end point. The first samples taken in the course of the illness showed a value of over 50 units in 42 out of 50 examinations. One patient, in whose serum no complement activity at all could be demonstrated, died two days later. In six patients, who were tested shortly before a recrudescence, the serum complement had increased before the appearance of symptoms. There was no indication of a relationship between increased serum complement activity and exhibition of salicylates. The authors discuss the possibility of the relationship between the rheumatic state and the allergic state, and the significance of the results obtained.

Congenital Antiviral Immunity in Swiss Mice.

JOHN A. ANDERSON AND VERN BOLIN (*The American Journal of Hygiene*, September, 1949) have studied congenital antiviral immunity in Swiss mice. Female white mice were vaccinated subcutaneously with a sublethal dose of murine encephalitis virus which was given daily for sixteen days; then a tenfold increase was begun. After one hundred and ninety-six days the animals could withstand relatively enormous doses of virus. After three months the females were bred and the litters weaned. Virus was added to the drinking water in known amount, and calculations were made of the volume of virus consumed per mouse per day. In the control mice from unvaccinated mothers there was a 98·8% mortality within twelve days. In the test group, that of animals born of vaccinated mothers, the experiments continued much longer, because the animals did not die. Five litters had no deaths, and amongst the rest there was an overall mortality of 21·6%. Some females had litters subsequent to the cessation of the immunizing injections, and these offspring withstood a larger dose of virus than those from non-vaccinated mothers. The authors suggest that the challenged, congenitally immune offspring mice lived longer and probably developed an active immunity to the virus of murine encephalitis.

Electron Microscope Study of Nerves Infected with Poliomyelitis.

E. DE ROBERTIS AND F. O. SCHMITT (*The Journal of Experimental Medicine*, October, 1949) have made an electron microscope study of nerves infected with human poliomyelitis virus. This followed establishment of the fact that the cut end of a nerve placed in a suspension of active virus was followed by paralysis of the corresponding muscle and lesions of the corresponding nuclei in the central nervous system. A similar technique of infection was used for the present series of experiments, the nerves being removed into cold Ringer's solution; then sections were cut and the fragments teased and prepared for electron microscopy. The uninfected nerve from the opposite limb was used as a normal control. A study of the pictures obtained showed that uninfected nerves had a relatively clear axial structure with periodic cross-banding in the neurotubules. Infected neurotubules showed small dense particles of from 160 to 500 Å, distributed along the axial walls in areas varying according to the length of time from the exposure of the cut nerve end to the virus, so that an "area map" of the granules could be constructed; after three hours' exposure to virus they were present only close to the cut end; after twenty-four hours they were a considerable distance away, a fact suggesting that the particles travelled a distance of approximately two millimetres per hour. In view of the difficulties of interpretation of these pictures, the authors thought it wise to state that the particles appear to be associated with virus infection, but no further characterization is possible at this time.

Production of Fever by Influenzal Viruses.

R. R. WAGNER, I. L. BENNETT AND V. S. LEQUIRE (*The Journal of Experimental Medicine*, October, 1949) have observed factors influencing the febrile response to single injections of virus in the beginning of a study on the production of fever by influenzal viruses. They used rabbits trained to handling and the use of the rectal thermometer, and took different groups of rabbits for each test. They found that a single intravenous injection of virus, either PR8 or Lee, was followed by a temperature rise of 3·5° C. within six hours; the temperature returned to normal within twenty-four hours, Lee showing a longer period of fall than PR8. Concentration of virus and suspension in saline also produced fever. When suspensions of virus were heated, if the hæmagglutinating capacity was lost, the fever-producing capacity was also lost; and similar results occurred if the virus was mixed with immune serum. An association of the occurrence of lymphopenia with fever was noted; the controls which did not have fever also did not have lymphopenia. If the virus was adsorbed onto red cells, heated so that the virus could no longer be eluted, then again the fever did not occur. It was concluded that the fever-producing quality was closely associated with the virus particle possessing the capacity to agglutinate red cells, and was heat labile with the hæmagglutinin. Contrasts were made with bacterial pyrogens, which do not disappear with the bacteria and are heat stable.

The same authors (*ibidem*) continue with a study of tolerance in rabbits to the pyrogenic effects of influenzal viruses. They state that this time they produced the fever with intravenous injection of virus and then challenged the animals with another injection next day, using either the same or a heterologous virus. Other groups of animals were left four, seven or eleven days, and at the end of this time the rabbits responded with fever as would normal rabbits. Estimations of antihæmagglutinin, however, showed

that the antibody production had continued to a peak, indicating that the fever had nothing to do with antibody. The lymphopenia disappeared in eleven days, and reappeared after the challenge dose. Heterologous viruses produced results in conformity with their position on the "receptor gradient" described by Burnet. Attempts were made to produce tolerance with bacterial pyrogens, and then to challenge with virus, but no tolerance could be induced. Blocking of the cells of the reticulo-endothelial system did not modify the resistance of the animals to a second injection. The authors discuss the possibility that the union of the virus particle with a receptor substance may play a part in the production of fever.

Increased Mortality following Penicillin Therapy.

G. E. FOLEY AND W. D. WINTER, JUNIOR (*The Journal of Infectious Diseases*, December, 1949), have studied the increased mortality following penicillin therapy of chick embryos infected with *Candida albicans stellatoidea*. The study was provoked by two instances of post-mortem isolation of the organism in chronic pulmonary infections in which penicillin treatment had been used over some period of time. Saline suspensions of the *Candida albicans* cultures were inoculated onto the chorio-allantoic membranes of ten-day chick embryos. Half the eggs received, in addition, 500 Oxford units of penicillin, the control eggs being given saline in order to maintain the dilution levels. The death rate was much higher in the eggs receiving penicillin than in the untreated eggs. Experiments with rabbits also showed that the animals infected by intravenous injection with subsequent penicillin therapy died within four days, while the control animals not receiving penicillin died after nine and sixteen days. The authors discuss the possibility that antibiotics may have a stimulating effect on *Candida albicans stellatoidea*.

HYGIENE.

Attack Rates Among Immigrants to Infected Populations.

J. IPSEN (*American Journal of Public Health and The Nation's Health*, February, 1950) demonstrates the effect of a reservoir of infection (scarlet fever patients in an infectious diseases hospital) on a group of "susceptible immigrants" (student nurses). It was shown that the incidence rate of scarlet fever and tonsillitis among student nurses at an infectious diseases hospital was four times that among nurses in a corresponding general hospital. Other factors affecting the incidence rate were length of service in the infectious hospital and seasonal conditions. An analysis of the independent influence of random sampling, community dosage, seasonal conditions and length of service shows the two last-mentioned factors to have, in this particular case, no significant effect on the variation in incidence rate. The relation of the incidence rate among the student nurses to the number of patients in the reservoir of infection follows an "S"-shaped curve similar to the "S"-shaped dosage-mortality curve commonly observed in bio-assays in which graduated doses are used. It demonstrates that the incidence rate does not increase proportionately to the reservoir of infection. A relatively greater increase in the incidence rate occurs when the reservoir of infection is greater. The author suggests that this may explain why the incidence in epidemic periods of a disease has a much higher ratio to the reservoir of infection than the ratio of new cases to the number of infective cases in endemic periods with a low reservoir of infection.

Porphyrins in Occupational Diseases.

C. C. MALOOF (*Archives of Industrial Hygiene and Occupational Medicine*, March, 1950) considers that the finding of coproporphyrin in the urine of those working with lead or lead compounds is an aid in the prevention or early diagnosis of lead poisoning. Coproporphyrinuria is not specific for lead poisoning, however, and must be used in conjunction with lead analyses. In the majority of cases investigated by the author it appeared earlier than red-cell stippling. The author suggests that this simple, rapid, economical test could be used to great advantage in the regular examination of workers in lead industries. Because of its simplicity it could be carried out as a routine during the time that elapses between the urinary analyses.

Fly Control Techniques.

J. H. COFFEY AND P. P. MAIER (*American Journal of Public Health and The Nation's Health*, May, 1950) consider that the control of flies is a legitimate function of health departments in the control of diarrhœal diseases and dysentery. They describe an experiment in which chemical fly control was carried out in a small group of cities in the county of Hidalgo, United States of America. In this county flies were prevalent and there was a high rate of infection with diarrhœal diseases. During the two years of the experiment there was a significant reduction in the mortality, morbidity and infection from diarrhœal diseases in the group of cities treated with DDT. Fly control was stopped in this group of cities and another group was treated. Diarrhœal diseases increased again in the untreated group and decreased in the second group of treated cities. The authors consider that these experiments reasonably prove that, where disease organisms are readily accessible to flies and fly populations are heavy, transmission of disease by flies actually occurs in nature. The authors consider that the control of flies by chemical means alone is not economical at present. The following procedures to bring about a reduction of flies are then described: (*a*) screening, (*b*) application of residual chemical sprays to selected fly resting surfaces, (*c*) application of larvicides in selected sites, (*d*) application of space sprays over extensive areas, (*e*) improvement of sanitation practices. The authors state that contact between human beings and flies is most effectively reduced by screening. Homes, food markets, restaurants and eating places should be screened. Residual treatment with DDT, methoxychlor or chlordane of all places where flies tend to congregate, for example, refuse bins and objects in the immediate vicinity, is the most effective and economical way of controlling flies with chemicals. Chlordane in the form of a 2% to 4% solution applied at the rate of 50 to 100 milligrammes per square foot to the surface of a larval breeding area will be reasonably successful in controlling fly larvæ. Space sprays from aeroplanes or ground-operated equipment are used to kill adult flies. The authors consider that the most effective space spray is a 5% DDT solution distributed in the form of droplets from 20μ to 100μ in size at the rate of one gallon to the acre. They state that control of flies by chemical means is only temporary, and in their opinion, the only sensible approach to the control of flies on a community-wide basis is improved environmental sanitation. The ultimate solution appears to be the development by the sanitary engineer of a complete system to provide for the sanitary disposal of putrescible wastes from their point of origin to their complete disposal.

Triethylene Glycol Vapour for Aerial Disinfection.

W. LESTER, S. KAYE, O. ROBERTSON AND E. DUNKLIN (*American Journal of Public Health and The Nation's Health*, July, 1950) have found that the vapour of triethylene glycol is the most suitable chemical compound to use for a bactericidal effect on airborne organisms. It is odourless, tasteless and extremely hygroscopic. When used in aerial disinfection it has no effect on fabrics or surfaces. Because of its low vapour pressure it is usually vaporized by heating. No evidence of toxicity was obtained from animal experiments carried on for more than a year. The amounts necessary for bacteriological action in the air are so minute that the quantity of triethylene glycol retained by inhalation never approaches a toxic level in humans. A method of determining the triethylene glycol content of the air is described. The authors state that the production of a fog indicates that excessive vaporization of glycol has taken place, but a slight fog indicates that sufficient amounts of glycol vapour are present. The humidity of the air controls the saturation concentration of the glycol vapour according to physico-chemical principles. At high humidities the airborne particles contain so much water that high bactericidal concentrations of glycol are unobtainable. Maximum rates of bactericidal action occur at humidities between 20% and 50% and with concentrations of glycol vapour above 50% saturation. At a constant saturation level of glycol vapour the rate of bactericidal action increases approximately twice for every rise of 15° F. between the temperatures of 55° and 85° F. To maintain a constant bactericidal concentration the rate of vaporization of the ethylene glycol must be sufficient to make up for the loss of vapour due to ventilation. Certain features of design applicable to suitable vaporizing units are described, and stress is laid on the avoidance of conditions leading to decomposition of liquid triethylene glycol. The oiling of floors and bed clothes and the maintenance of high standards of personal hygiene are indicated as desirable adjuncts to the use of triethylene glycol vapour for aerial disinfection.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Repatriation General Hospital, Springbank, on June 22, 1950, DR. C. O. F. RIEGER, the President, in the chair. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Suppurative Pneumonitis.

DR. C. D. SWAINE presented a patient who had been admitted to hospital on April 1, 1950, with a history of gradual onset of ill health over a period of two months. He had lost one and a half stone in weight in the three months preceding admission, had a poor appetite and had felt considerable loss of energy. In the evenings he had been feverish with a severe headache. He also complained of pain in the left upper part of the abdomen, which radiated through to the back, cough, with one or two ounces of yellowish sputum, most noticeable in the mornings, and night sweats. There were no symptoms referable to his alimentary or genito-urinary system, and he was not dyspnœic. On physical examination he was a tall, asthenic individual, who had obviously lost weight. His blood pressure was 135 millimetres of mercury (systolic) and 85 millimetres (diastolic). A patch of tubular breathing was audible over the second right intercostal space anteriorly. No other abnormal physical signs were found. Blood examination revealed a hæmoglobin value of 16·2 grammes per 100 millilitres, a total white cell count of 7100 per cubic millimetre, made up of neutrophile cells 65%, lymphocytes 26%, monocytes 7%, eosinophile cells 1% and basophile cells 1%, and a blood sedimentation rate of 17 to 28 millimetres (Westergren). Twelve specimens of sputum were examined and no acid-fast bacilli were found; but the sputum contained numerous pus cells, and on culture a scanty growth of diphtheroid bacilli was isolated (culture was repeated twice). The Mantoux test yielded a positive result (0·00002 gramme of P.P.D.). On bronchoscopic examination the only abnormality found was slight redness of the mucosa on the right-hand side. X-ray examination of the chest and a bronchogram showed a lesion progressing from lobar consolidation at first to a shrunken, cavitated bronchiectatic lobe. A tomogram showed no definite evidence of cavitation, though in one cut a very suspicious area was seen. The patient was treated with penicillin and aureomycin without any great benefit.

Dr. Swaine said that the case had been reviewed by members of the hospital chest division after the taking of a bronchogram on June 8, 1950. Bronchograms were studied, and it was thought that they showed the presence of a mild grade of bronchiectasis in the auxiliary branches in the upper lobe of the right lung. There was also a considerable portion of that lobe into which none of the lipiodol had entered. The case was discussed with regard to all the findings, and it was thought probable that there existed, in the upper lobe of the right lung, small abscess cavities, to which the dilatation of the bronchial tubes was merely secondary and that removal of the lobe offered the best chance of complete recovery. In reviewing the case at the time, Dr. Swaine had referred to an article by Sellers, Thompson, Houghton and Blair in *Thorax* of July, 1946. He listed the diseases which it was necessary to exclude before the making of a diagnosis of non-specific suppurative pneumonitis and indicated where he considered that the disease fitted into the classification of suppurative pulmonary diseases given by the authors quoted. Dr. J. G. Sleeman, in opening the discussion, had said that he would consider the diagnosis more definitely established if the presence of cavitation had been proven by the special X-ray techniques employed. However, he felt that, as other diseases seemed to have been excluded, the diagnosis of non-specific suppurative pneumonitis was the best which could be made on the evidence available. The diagnosis having been made, the treatment of choice was lobectomy. Dr. H. D. Sutherland had said that he agreed with Dr. Sleeman's views and commented that the absence of the phenomenon of spreading was probably accounted for by the fact that the patient had been receiving large doses of antibiotics. Dr. Sutherland agreed that lobectomy was the treatment which offered the patient the best chance of recovery.

Dr. Swaine went on to say that before the operation was proceeded with a lateral tomogram was obtained. That, also, was inconclusive. On July 13, 1950, lobectomy was performed. The following account was given of the pathological findings:

Upper Lobe of Right Lung.—When fixed the lobe is 17·5 cms. long, 11·0 cms. wide and 6·5 cms. deep. There is considerable tuberculous infiltration of the upper two-thirds of the specimen, and ending at its lower margin along a sharply defined line, 1 cm. below the hilum on the medial aspect and 3·5 cms. below the hilum on the dosal aspect.

The involved tissue shows lobulated and circular areas of caseation, especially on the upper medial aspect adjacent to the hilum and becoming irregular laterally, with numerous peripheral miliary nodules which extend further caudally on the lateral aspect.

Continuous with these lobulated areas of caseation medially and the largest irregular area laterally is an area 4 cms. × 2·5 cms. × 2·5 cms. which is cream-coloured, friable, necrotic tissue, and which projects slightly above the dorsal surface opposite and a little above the hilum.

On the lowest outer angle of the lobe are several large emphysematous bullæ up to 2 cms. in diameter and containing a framework of fibrosed pulmonary tissue.

At the apex, there is a peripheral zone, 1 cm. deep, of similar emphysematous tissue, with a bulla, 2·5 cms. in diameter, at the lateral aspect and with fibrosed walls lined by pale caseous material, but still containing trabeculæ as above; this appearance simulates a large cavity.

Microscopic examination of sections from the dorso-lateral aspect of the lobe shows extensive tuberculosis. Sheets and nodules of tissue show marked infiltration by epithelioid cells, fibroblasts and giant cells, with less lymphocytes and plasma cells, and relatively small areas of caseation. Acid-fast bacilli morphologically resembling Myco. tuberculosis can be seen.

Confluent "solid" tubercles, tuberculous granulation tissue and the relatively small amount of caseation are the main microscopic features of this specimen.

(A further report on the bronchus and the "cavity" will be forwarded after paraffin imbedding.)

Diagnosis: pulmonary tuberculosis.

Polyarteritis Nodosa.

DR. D. L. WILHELM discussed a case of *polyarteritis nodosa* and presented autopsy specimens. The patient, a man, aged twenty-seven years, had been admitted to hospital on December 12, 1949, for review, as he had suffered from asthma for three to four years previously. On admission, he had a pink macular rash over the forehead and behind both ears. The liver edge was palpable below the right costal margin. Each evening his temperature rose to 101° F., his pulse rate was 100 beats per minute and his respiratory rate 20 per minute. Throughout his illness the temperature rose in the late afternoons to 100° to 101° F. Examination of his blood revealed a hæmoglobin value of 14·5 grammes per 100 millilitres, a total white cell count of 14,500 per cubic millimetre, and a differential count of neutrophile cells 52%, lymphocytes 29%, monocytes 2%, eosinophile cells 17% and basophile cells nil. An X-ray examination of the chest showed that the right lung was less translucent than the left and suggested consolidation below the transverse fissure with pleural involvement. After two weeks' Christmas leave the patient was readmitted to hospital on January 4, 1950. He had a pleural effusion at the base of the right lung; the fluid contained 4000 leucocytes per cubic millimetre, of which 95% were neutrophiles, but no growth was obtained on attempted culture. During the next few weeks a number of investigations were performed and repeated. The result of a Mantoux test was negative. In examination of the sputum, acid-fast bacilli were not detected. Guinea-pig inoculation with the pleural effusion did not reveal evidence of tuberculosis. Bronchoscopy was inconclusive. The results of the Casoni and hydatid complement-fixation tests were negative. The serum sodium content was 350 milligrammes per 100 millilitres. The results of attempted blood culture and sputum culture and of agglutination tests were negative. A blood count on January 12 showed that the white cell count had risen to 18,000 cells per cubic millimetre, and in the differential count, eosinophile cells formed 28% of the leucocytes. Penicillin therapy was commenced, 100,000 units being administered four-hourly. That dosage was subsequently increased, and aureomycin and sulphadiazine were administered also. By February 28 the pleural effusion was bilateral; the hæmoglobin value had fallen to 11·0 grammes per 100 milli-

litres, the white cell count was 20,000 per cubic millimetre and eosinophile cells formed 41% of the leucocytes. Blood platelets numbered 150,000 per cubic millimetre. The blood picture did not change subsequently, and sternal puncture gave no further information. Microscopically the urine showed one or two leucocytes and four to five erythrocytes per high-power field. That result was obtained many times up to within two days of death. On March 30 the provisional diagnosis was (i) *polyarteritis nodosa* and (ii) Hodgkin's disease. Within four days a more obvious and tender subcutaneous nodule was found over the fifth rib in the right anterior axillary line. Previously, several doubtful and small subcutaneous nodules had been palpated in the anterior abdominal wall. The fresh anterior axillary nodule was excised for biopsy, and a diagnosis of *polyarteritis nodosa* was made. Subsequently the eosinophile cell index was inconclusive, the non-protein nitrogen level was 25 milligrammes per 100 millilitres, and the serum sodium level 325 milligrammes per 100 millilitres. The patient was treated from April 18 with testosterone five milligrammes given intramuscularly and ascorbic acid 1·0 grammes given intravenously twice weekly. His condition regressed and he died on May 10.

In summary, Dr. Wilhelm said that the patient was a male subject, aged twenty-seven years, with a previous history of asthma; he had eosinophilia, subcutaneous nodules on the trunk and bilateral pleural effusion. Discussing eosinophilia and subcutaneous nodules, Dr. Wilhelm said that Logue and Mullins, writing in 1946 in *Annals of Internal Medicine* (Volume XXIV, page 11), had tabulated the clinical features in 177 cases of *polyarteritis nodosa* and had emphasized the fact that eosinophilia and subcutaneous nodules were not necessarily present alone or in combination in *polyarteritis nodosa*; they reported eosinophilia in 35% of cases and nodules in 23%. Tonkin and Pulvertaft, writing in 1948 in *The Lancet* (Volume II, page 291), had described skin nodules and eosinophilia in less than 50% of their cases. With regard to pleural effusion, Dr. Wilhelm had been unable to find any reference to it in the literature as a feature of *polyarteritis nodosa* at any stage of the disease. Discussing hæmaturia as a feature of the disease, he said that not more than four or five red blood cells per high-power field had been found in the urine at any stage of the disease, although the kidneys were involved according to microscopic examination. Harris *et alii* in 1939 had reported in *Archives of Internal Medicine* (Volume LXIII, page 1163) the demonstration of renal lesions in 87% of cases at autopsy. Logue and Mullins had reported hæmaturia in half of their series, but stated that pathologically the kidneys were involved in 80% of cases.

Dr. Wilhelm demonstrated the macroscopic and microscopic features of autopsy specimens. In the myocardium at the extreme apex of the left ventricle there was a hæmorrhagic and necrotic round focus one centimetre in diameter, bounded by a wall of fibrosis and extending to the endocardial surface. Similar smaller foci one to three millimetres in diameter were scattered in the myocardium on the anterior aspect of the apex of the left ventricle. The remainder of the left ventricle anteriorly was considerably fibrosed. There was ante-mortem thrombus attached to the wall of the right auricle, with dense pericardial adhesions. In the right pleural space were 75 ounces of straw-coloured fluid, with 65 ounces on the left side. Densely thickened pleura covered the right lung and deformed the upper lobe of the right lung. The right lung was firm and tough from carnification, and both lungs contained multiple infarcts of different ages. The kidneys had a slightly granular surface, and the small bowel contained multiple small mucosal ulcers. There was venous congestion of the liver and pancreas. Discussing the microscopic pathological features, Dr. Wilhelm pointed out that *polyarteritis nodosa* was an obliterative, inflammatory vascular disease involving the small arteries and arterioles and characterized by fibrinoid necrosis of the vessel walls. The older name of periarteritis was undesirable because all coats were involved. The disease was uncommon, and males were affected more commonly than females. It might occur at any age, but 50% of the patients were in the fourth and fifth decades. The organs most frequently attacked were the kidneys (80%), heart (60%), liver (50%), spleen, lungs, mesentery, peripheral nerves, skin and brain. The lesions were irregular in distribution and showed four pathological stages, according to Arkin, writing in 1930 in *The American Journal of Pathology* (Volume VI, page 401). The first stage was that of fibrinoid necrosis, which was most prominent in the media; in the larger vessels the necrosis began in the outer portion of the media; in the small arterioles it commenced adjacent to the internal elastic membrane. In the second or acute inflammatory stage, the coats became infiltrated with neutrophile and eosinophile cell,s lymphocytes and plasma cells. In the third stage granulation tissue was laid down in all coats according to their involvement. The fourth stage was that of healing; the wall was replaced by scar tissue and periarterial fibrosis, and the lumen was partly or totally occluded. All coats of the affected vessels were involved, and as a result of their occlusion by intimal proliferation, thrombosis with infarction occurred. Also, aneurysmal dilatation and nodule formation were common and might give rise to the "peas in the pod" appearance. In the case under discussion the necrotic, acute inflammatory and granulation stages were demonstrated in kidneys, pancreas, testis and myocardium. The healing process was well illustrated in the cardiac lesions and in a section of pectoral muscle. Dr. Wilhelm said that it was of interest to note how frequently various authors had reported the close association of *polyarteritis nodosa* and rheumatic fever. In the case being discussed, if the well-defined necrotic foci were not present in the heart muscle, the myocardial granulation tissue and fibrosing lesions would serve well to illustrate healing of gross rheumatic heart disease.

In conclusion, Dr. Wilhelm said that he would like to repeat the warning of Harris (1939) that *polyarteritis nodosa* would be missed clinically, if it was not searched for. Hæmaturia, eosinophilia and subcutaneous nodules were not always present together. The diagnosis should be considered in cases of unexplained hæmaturia with fever, disproportionate tachycardia, peripheral neuritis, cardiac arrhythmia or a changing electrocardiogram, persistent moderate polymorphonuclear leucocytosis, recurrent polyarthritis, abdominal pain or transitory pulmonary infiltration. Biopsy should be performed immediately on suspected nodules, for even a few days' delay might mean loss of the opportunity of microscopic confirmation.

Leprosy of Lepromatous Type.

DR. R. V. SOUTHCOTT discussed a case of leprosy of lepromatous type. Owing to transport difficulties it was not possible to present the patient in person (he was a patient in the Northfield Infectious Diseases Hospital), but photographs exhibiting the leonine appearance and skin nodules were presented. It was pointed out that in the last twelve months two cases of leprosy had been diagnosed at the Repatriation General Hospital. The first was a case of neural type in a man who had lived for a number of years at Tennant Creek in the Northern Territory, and who was believed to have contracted the disease from a tent-mate there. The second case was the one reported at the meeting. The patient was aged sixty-one years at the time of the meeting. He had been admitted to the skin ward of the Royal Adelaide Hospital on October 29, 1946, suffering from blisters on the arms and legs, below the elbows and knees, which had occurred for two years; they would come up overnight, enlarge, and burst. He had suffered from an occasional attack of malaria in the Darwin region. He had lived in the Northern Territory for many years. Examination revealed large pustular vesicles on the legs, which were red, hot and œdematous. There were coppery-coloured scaly patches on the skin of the abdomen. He was considered to be possibly suffering from a streptococcal infection of the arms and legs. He was treated with zinc cream and eusol compresses, and later with penicillin cream.

On November 28 and December 12, 1946, Wassermann tests yielded "doubtful positive" results, and on December 23 a course of 12 injections of "Acetylarsan" was started. On January 11, 1947, the patient was still complaining of numbness of the hands and feet, a Kline test on January 23 yielded a doubtful positive result and on January 29 he was considered to be suffering from *tabes dorsalis* and was transferred to a medical ward. He was found to have loss of sensation to pinprick over the forehead and nose, upper part of the arms and stocking area of the legs, with no pain sense in the *tendo Achillis* and no vibration sense in the tibia. The note was made on his records on March 29, "Tabetic neurological symptoms as before", and he was discharged from hospital to home. On July 28, 1948, the ulcers had recurred on the legs, he complained of a sensation of pins and needles in the hands and feet, and was readmitted to the skin ward of the Royal Adelaide Hospital. On August 12 the possibility of leprosy was considered and rejected; the result of a Wassermann test was at that stage negative. In December the absence of eyebrows was noted, the patient was considered to be myxœdematous, and a course of thyreoid tablets was instituted. On January 9, 1949, he became cyanosed, and the œdema of the feet increased. On January 31 a discharging sinus was present in the left buttock. The result of a Wassermann test on February 18 was negative. Reexamination of the neuro-

logical system revealed diminished sensation in the hands and feet, and subsequently anæsthesia of the palate was recorded, being considered as possibly due to hysteria. The dermatologist recorded the skin condition as cured. On April 5 the sinus in the buttock was excised surgically, and subsequent section showed "fibrocaseous tuberculosis with typical tuberculous giant cells". On April 14 examination of gastric contents revealed acid-fast bacilli; some of the fluid was injected into guinea-pigs. On April 20 the wound in the buttock had broken down and again leprosy was suggested as a possible diagnosis. A swabbing from the buttock wound revealed acid-fast bacilli. In June the guinea-pigs were sacrificed. Examination of the first revealed no abnormality, but the second was reported as being "a generalized infection with tuberculosis". X-ray examination of the chest on July 20 revealed no abnormality, and the patient was considered to be suffering from tuberculosis of the skin, but not of the lungs. In August an acute attack of abdominal pain occurred, and the blood pressure was much reduced. An electrocardiogram revealed no abnormality, but later T_1 and T_4 became depressed, and then inverted. A diagnosis of anterior myocardial infarction was made. On March 7, 1950, the patient was transferred to the Repatriation General Hospital, Springbank, with a diagnosis of myocardial degeneration and *tabes dorsalis*. Examination of the patient there revealed a leonine facies, with complete loss of eyebrows except for two hairs on one side. There was a diminution of light touch sensation over the lower part of the arms and legs, and temperature discrimination was greatly reduced in the hands and feet. The knee jerks were active and equal; the ankle jerks were weak and equal; the plantar responses were flexor. There was no neural thickening and no Rombergism. The results of blood Wassermann, Kline and Berger-Kahn tests were negative. Acid-fast bacilli were present in the sputum. Chest X-ray examination revealed no abnormality. Examination of a nasal smear showed extracellular clusters of acid-fast bacilli, decolorized after forty minutes in 5% sulphuric acid solution. The result of the Mantoux test was positive. The results of Wassermann, Kline and Berger-Kahn tests of the cerebro-spinal fluid were negative. The ear, nose and throat surgeon reported that the patient had "clinically a typical nasal lues with perforated septum and collapse of bridge". Guinea-pigs inoculated with gastric washings were sacrificed, but no abnormality was detected in them. On April 4 the pathologist's report on the examination of a nodule from the skin of the back was received, the clinical, pathological and bacteriological features of the case were reviewed, and the diagnosis of leprosy was established. Public health action was instituted, and treatment for leprosy commenced.

Dr. Southcott pointed out that the diagnoses of syphilis, leprosy and tuberculosis had all been made in the one case and suggested that the clinical and pathological features which had led to those three diagnoses were all due to the one disease, leprosy. He said that positive Wassermann reaction occurred in about 50% of all cases of leprosy, being rather commoner in lepromatous than in neural leprosy. How many of those positive reactions were due to concomitant spirochætal infection was a matter of some argument. The discharging sinus which was described as tuberculous he associated with leprosy rather than tuberculosis. The reaction in leprosy was frequently a tuberculoid one, with a pathological picture indistinguishable from that of tuberculosis. The infection of one of the guinea-pigs with tuberculosis was not satisfactorily accounted for, as three other guinea-pigs similarly inoculated showed no infection. There did not appear to be adequate grounds for a diagnosis of *tabes dorsalis*. In many cases of lepromatous leprosy minor neurological changes were found on the sensory side. It was thought that some of the difficulty in the diagnosis was due to the patient's suspecting the diagnosis, and being fearful of incarceration in a leprosarium. He was very ready to attribute his peculiar facial appearances to overmuch boxing in his youth. He admitted to having mixed freely with natives in the years that he spent droving cattle in the Northern Territory.

DR. D. L. WILHELM presented microscopic sections which had been prepared from the nodule excised from the middle of the patient's back. Sections stained with hæmatoxylin and eosin showed typical lepromatous leprosy. The subepithelial connective tissue was increased in amount and contained numerous irregular columns and cords of chronic granulomatous tissue with mononuclear and epithelioid cells, fibroblasts, lymphocytes, plasma cells and occasional small giant cells of the foreign-body type. With Ziehl-Neelsen staining, numerous acid-fast bacilli were demonstrated, confined within the cords of granulomatous tissue. The acid-fast bacilli were found within "foamy" mononuclear cells or occurred as extracellular globi. Smears prepared from sputum and specimens obtained by gastric lavage contained numerous acid-fast bacilli, which frequently occurred in large clusters or bundles of bacilli.

DR. C. M. DELAND said that he was looking after the patient at the Northfield Infectious Diseases Hospital. He considered his condition a most obvious case of dermal leprosy. He preferred the division of leprosy into dermal, nodular, neural and tuberculoid forms to the international classification of lepromatous and neural forms. Dermal leprosy was seen most commonly in Europeans. He was treating the patient with "Moogrol" and "Diasone", and expected him to respond well to treatment. He regarded his age as the only unfavourable factor in the prognosis. It was proposed to treat him with the new antileprotic drug "Promacetin" as soon as it became available. The neural condition had also responded well to treatment. Dr. Deland regarded "Moogrol" as the most effective drug of the hydnocarpus derivatives available in the treatment of leprosy. He considered that it should be given to the limit if tolerated, the guiding factor being the presence of albuminuria; the maximal dose was then controlled by lengthening or shortening the intervals between doses. He thought that the positive result in the Wassermann test in the case under discussion indicated concomitant syphilis. The result became negative after treatment with arsenic. He thought it also likely that the patient was suffering from tuberculosis.

PROFESSOR J. B. CLELAND said that the fact that the acid-fast bacilli had been seen in clumps in the sputum and gastric washings had not been appreciated sufficiently; he thought that if that had been done the diagnosis could have been made earlier.

Dr. Southcott, in reply to Dr. Deland, said that so many mixed types of leprosy occurred that it was difficult to classify the cases accurately; he considered the simpler international classification more suitable for clinical use, public health and statistical purposes. The more extensive classification was that usually used for pathological purposes. The main point of the presentation was to bring forward a diagnostic problem, and to remind the audience that sporadic cases of leprosy in whites did occur in Australia.

Rheumatoid Arthritis.

DR. KENNETH SANDERSON presented three patients with rheumatoid arthritis to demonstrate the response to the combined injection of testosterone and ascorbic acid. He said that testosterone propionate, 10 milligrammes in oil, was injected intramuscularly; ascorbic acid, 10 millilitres of a 10% solution, was injected intravenously five to ten minutes later. The treatment was given daily for two weeks, and then twice a week or less, depending on the progress. Physiotherapy was given as energetically as the patient's condition would allow.

In the first case a very good result had been obtained. The patient, a commissioned officer in the permanent army, aged sixty years, had developed rheumatoid arthritis eighteen months before, and had been treated in the hospital by the usual measures with slow incomplete recovery. He was admitted to hospital again in January, 1950, because he had lost two stone in weight over the past six weeks and felt unwell, and the joints of his hands, wrists, elbows, left ankle and feet had become painful, and his neck had become stiff. Examination of the patient revealed evidence of rheumatoid arthritis of moderate degree. His blood picture and blood sedimentation rate were normal. X-ray examination of his sacro-iliac joints showed no abnormality. Over the next three months he showed a very gradual improvement with the usual measures of treatment. He was given his first combined injection treatment on March 21, 1950. The result was dramatic. He lost the pain in the joints within a very short period, the muscular power and range of movement increased notably, and the range of rotation of his neck increased from 55° to 110°. In the first fortnight he gained nearly 14 pounds in weight, and he had a feeling of well-being. At the time of the meeting he had been back on duty for two months and had been classified B2 by a medical board. He felt better than for two years and required treatment once a week or fortnight.

In the second case a good initial response occurred, but only partial restoration of function was obtained after three months' treatment. The patient, a service pensioner, aged fifty-one years, had developed acute arthritis in the left wrist four and a half years earlier, and subsequently his other wrist, hands, elbows, shoulders, knees, ankles and feet had been affected. His condition had fluctuated since then, but had remained crippling. He had had all the usual

treatment for rheumatoid arthritis. He was admitted to the hospital in February, 1950, and showed the changes of moderately severe rheumatoid arthritis. The blood picture was normal, the blood sedimentation test (Wintrobe) revealed a fall of one millimetre in the first hour, and X-ray examination of the hands showed advanced arthritic changes in the wrists and arthritic changes in the small joints. There were changes of chronic spondylitis in the lower part of the dorsal and the lumbar regions. For the first month of conventional therapy there was no improvement; he hobbled around with the aid of a walking stick. He was given his first combined injection on March 24 with dramatic results. Half an hour afterwards he walked briskly down the ward without his stick, and found all the affected joints less painful and more mobile and powerful. For the first fortnight he improved rapidly and gained 10 pounds in weight, but since then his progress had been much slower, and his condition had actually deteriorated recently while he was worried for a few days about his son's health. His blood sedimentation rate had risen to 25 millimetres in the first hour, 86 in the second.

The third patient, a service pensioner, aged fifty-nine years, had shown only moderately satisfactory response. During the first World War he had suffered from severe rheumatic fever, and though that had resolved and he had worked as a carpenter until two years before the meeting, he had suffered from fleeting pains in the various joints. He had then developed acute polyarthritis involving all the joints except the hips and spine. He had been treated in the Royal Adelaide Hospital for five months, and since then at the repatriation out-patient clinic. He had had all the usual forms of treatment, and although he had improved considerably, his joints had remained stiff and painful and his muscles weak. He was found to have a raised blood uric acid level and had been treated with colchicine. On admission to hospital in April, 1950, the patient had presented the picture of moderately advanced rheumatoid arthritis. His blood picture was normal. The blood sedimentation rate was 70 millimetres in the first hour and 114 millimetres in the second. X-ray examination of the feet showed changes in the joints consistent with rheumatoid arthritis. His blood uric acid level was 4·6 milligrammes per 100 millilitres. Treatment with combined injection was begun on April 11; his pain had decreased and his joints were a little more mobile. He considered that treatment had been worth while, but the objective evidence of improvement had not been remarkable.

In discussing these three patients Dr. Sanderson said that he had purposely selected patients showing a favourable response, not because he wished to produce an exaggerated impression of the treatment, but because he wished to demonstrate that it was of value in selected cases. An equal number of patients who showed no response could just as readily have been presented. He hoped that members would leave the meeting with a feeling of guarded optimism for the treatment as an adjunct to the well-established measures of improving the general health, correcting deformities and restoring the function of affected limbs. Though it was cruel to delude patients with a disease such as rheumatoid arthritis into false hopes about a new method of treatment, it was even more cruel to withhold such treatment if it could do no harm and might do good.

Dr. Sanderson went on to say that the primary object of treating the disease at Springbank had been to restore the efficiency of the patient as quickly as possible. Controlled assessment of the value of the treatment would soon be forthcoming from large centres elsewhere. The reports in the medical Press had so far been very divergent. A recent copy of the *British Medical Journal* had contained a letter from 12 prominent rheumatologists who stated that they had obtained no benefit from the treatment in 172 cases. Though their opinions must be given considerable weight, it would be wise to defer final judgement until they had presented a detailed report of their material and methods. The selection of patients to be treated and the method of treatment were of equal importance if good results were to be obtained from any form of therapy. With respect to selection of patients, Dr. Sanderson said that only impressions could be given at present. The best results seemed to be obtained with conditions of relatively recent onset. The most dramatic result he had seen had been in a patient of Dr. Guy Lendon's at the Royal Adelaide Hospital. That patient was a young man, who had had acute extremely painful rheumatoid arthritis of the hands and feet for less than two weeks, and had obtained complete and lasting relief one and a half hours after the injections were given. The more advanced the periarticular changes, the less likelihood of improvement there seemed to be. In the individual patient those joints most recently involved responded best. The best method of selection was to give two or three treatments at daily intervals. A definite response within an hour or two of injection was a favourable omen; no response after three injections reduced the chances of improvement from the treatment to almost nil. Such a trial, if explained to the patient, neither raised false hopes nor caused undue expense.

As to the method of treatment, Dr. Sanderson said that he had followed that originally described with one important difference—the use of testosterone in preference to desoxycorticosterone. Those two substances and progesterone were all closely allied chemically, and early reports had indicated that all were apparently effective when used with ascorbic acid. If the treatment was to be used frequently with the idea of enabling the physiotherapist to work on joints with less pain and muscles with less spasm, desoxycorticosterone had the theoretical disadvantage of causing sodium retention. Testosterone caused only an insignificant retention of sodium and had the further advantage that it seemed to be more intimately concerned in muscle metabolism than desoxycorticosterone. For those reasons testosterone was chosen as the steroid of preference to be used in men. No side effects had been noticed, and the results obtained appeared to be as good as those when desoxycorticosterone was used. Dr. Sanderson said that he did not feel qualified to comment on the biochemical or endocrine aspects of the treatment. Other observers had suggested that the ascorbic acid reacted chemically with the steroid to produce a cortisone-like substance, and that just as good results could be obtained by replacing ascorbic acid with methylene blue. There was also evidence that if such a reaction occurred it did so in the peripheral tissues, not in the adrenal glands. The clinically observed facts that the blood sedimentation rate and the number of circulating eosinophile leucocytes did not change confirmed that observation. Any explanation of the action had to account for the very rapid relief of pain and increase in movement. Rheumatoid arthritis was a disease of periarticular tissues, of ligaments and tendons, and when the onset was acute, it was obvious that an alteration in the vascular equilibrium had occurred, causing local swelling of periarticular tissues with pain and reflex muscle spasm, and possibly synovial effusion. Even in chronic arthritis the temperature, colour and moistness of the palms and soles attested to the importance of local vascular disturbance. An additional fact that Dr. Sanderson had noticed was that patients with cold cyanosed hands sometimes had surprisingly red blood when venepuncture was performed, suggesting (as Professor Wood Jones had recently pointed out for gout) that an abnormal arterio-venous anastomosis was present. It seemed reasonable to assume that, whatever the biochemical changes might be, the physiological effect of the treatment was to alter the hæmodynamics in the affected tissues, reducing the swelling and thereby relieving pain and muscle spasm. There was no doubt that in some cases a feeling of warmth in the skin followed rapidly after the injection of ascorbic acid, while at the same time the sensation of heat in the inflamed joints disappeared.

DR. N. S. GUNNING said that he had treated three patients by the method described, and he had been impressed by the state of well-being that had followed. A patient with ankylosing spondylitis who had been under treatment with deep X-ray irradiation had shown a general improvement, both in his physical state and in his attitude to his illness. A patient with rheumatoid arthritis had been able to move his fingers much more freely. Dr. Gunning said that he had been told that the treatment might have a damaging effect upon the pituitary gland, and wondered whether any other members present could give more details on that point.

DR. M. E. CHINNER said that he had used the treatment for two patients, and after six injections they had not improved. He wondered if the patients presented by Dr. Sanderson had shown such improvement because they were treated as bed patients in hospital. He had recently discussed the treatment with a Sydney physician who had used it for 30 patients. Among those there was improvement in only one case, and that was regarded as possibly due to psychogenic factors.

In reply, Dr. Sanderson said that he had not heard of any damaging effects upon the pituitary gland. He felt that the improvement shown by his patients could not be attributed to in-patient treatment, as they had all been ambulant. He stressed the importance of physiotherapy, splinting and improvement of the general health as the major therapeutic agents in rheumatoid arthritis, and said that the injections were mainly of value in facilitating the work of the physiotherapist.

Correspondence.

ACROPATHY, ACRODYNIA INFANTILIS (FEER), PINK DISEASE (SWIFT) AND THEIR DIFFERENTIAL DIAGNOSIS AS TO USTILAGINISM (MAYERHOFER).

SIR: I should like to comment on two articles on pink disease in your journal on January 28, 1950.

I. Terms and Definitions.—In 1938 I published a lengthy exposition on various "acropathies" in childhood in the *Ergebnisse der inneren Medizin und Kinderheilkunde*, Volume LIV. The Greek technical term "acropathy" was used in its widest sense covering processes and conditions "having an obvious tendency to develop special symptoms on extremities". For purposes of pædiatric literature, that is, for the theory of the clinic, I still adhere to the idea of acropathy as first mentioned—as a kind of "theoretical reserve", that is, for the logical construction of the scheme set forth in the next paragraph. Moreover, I chose another expression for the clinic, having a narrower meaning, that is, "complex of acrodynoid symptoms", which I defined as the morphological group of diseases being identical with, or related to, infantile acrodynia or which have, in medical practice, to be taken into consideration as possible differential diagnoses.

II. Clinical Scheme of Classification of All Acropathies Occurring in Childhood.—In 1938 I devised for clinical purposes the following classification which I still maintain:

Acropathies.

1. Infantile acrodynia (Selter, Swift, Feer, Debré, Péhu, Boucomont, Rocaz, Woringer): classical forms.

2. Infantile acrodynia *"formes frustes"* according to Mayerhofer and collaborators.

3. Complexes of acrodynoid symptoms: (*a*) Chronic arsenic poisoning with acropathic skin symptoms. (*b*) *Morbus cerealis* as a general group—(i) peliagra; (ii) secalism; (iii) ustilaginism (food poisoning of infants due to maize smut); (iv) more rare food poisonings due to unclean cereals (experimental basis by Debré and Névot, clinical observations by St. Dimitrijevic), *ustilago nuda forma tritici, Agrostemme Githago* with the poisonous saponin "githagin" *et cetera.*

4. General group of acropathic symptoms or syndromes for differential diagnosis: (*a*) Raymond's disease. (*b*) *Chorea fibrillaris* (Morvan). (*c*) *Acrodermatitis mutilans* (Parkes-Weber). (*d*) *Acrocyanosis chronica.* (*e*) Erythromelalgia. (*f*) Acropathic symptoms of purpura and scurvy. (*g*) *Keratoma hereditare palmoplantare* (dominantly hereditary). (*h*) *Keratosis extremitatum hereditaria progrediens* (Kogoj), or *"Mal de Meledda".* (*i*) *"L'acrodermatite continue suppurative"* (Hallopeau). (*j*) *Psoriasis pustulosa.* (*k*) Resistant pustular rashes on palms and soles. (*l*) Acrosclerodermies, dermatomyositides, *acrodermatitis atrophicans et cetera.*

III.—Etiologico-Pathogenetic Differences and Clinical Differential Diagnosis between Acrodynia Infantilis (Selter-Swift-Feer) and Ustilaginismus (Mayerhofer).—In July, 1939, I had the pleasure of exchanging correspondence with Dr. F. W. Clements, of the Australian Institute of Anatomy, Canberra, about the food poisoning of infants due to maize smut, which I had discovered shortly before (1929-1930) and about a new disease which I had called "ustilaginism". Dr. Clements wrote to me then that according to the view of some Australian pædiatricians the Australian "pink disease" is related to rust diseases of cereals. I think he was right in saying related and not identical. Unfortunately war and post-war conditions have prevented a further exchange of views. Recently, however, I found in THE MEDICAL JOURNAL OF AUSTRALIA (January 28, 1950) two interesting papers discussing, among other texts, also my two articles on ustilagoes. One of the purposes of this small communication is to come again into scientific relations with my two Australian colleagues. Moreover, I would like them to know that much interest has been aroused at the Pediatric Clinic in Zagreb by the two articles on pink disease (Donald B. Cheek and H. C. T. Stace and Donald B. Cheek and Stanton Hicks in THE MEDICAL JOURNAL OF AUSTRALIA, January 28, 1950, pages 101-122). Dr. Očko, a lady assistant at the clinic, reported very comprehensively about these two important papers in a monthly meeting of the pædiatricians' section (on April 28, 1950). We were very interested to learn about the new treatment (Cheek and Hicks) with simple sodium chloride in connexion with desoxycorticosterone.

A further important purpose of this letter is the clearing of a misunderstanding. In the aforesaid publication by Cheek and Stace I found on page 103 the following passage: "Mayerhofer (1938) put forward that the disease might be due to spores of a cereal smut, which contaminated bread. This question was gone into in Australia by Clements (1940) and he showed the claim to be invalid."

This text might imply that I myself had confounded the food poisoning due to maize smut spores ("ustilaginism") with the "pink disease" (Australian variety of the *acrodynia infantilis*, type Swift). But precisely this is not the case. Right from the beginning I represented my ustilaginism as a special syndrome, even as a "new disease" which is ætiologically entirely different from *acrodynia infantilis.* I tried myself to characterize the symptoms of ustilaginism by the catchword "fire" (no perspiration, dry decrustation on hands and soles) and those of *acrodynia infantilis* (Feer) by the catchword "water" (free perspiration, moist maceration scales on hands and soles). Thus I desired to point out clearly and precisely the extreme differences in the symptomatology of the two diseases, that is, of *acrodynia infantilis* and of my ustilaginism.

IV. No Unanimity in European Literature.—In European literature there is no doubt about the independence of ustilaginism in so far as it is mentioned at all. Feer himself accepted my thesis and arguments for the independence of my ustilaginism's appearance and wrote to me in April, 1938, that he pointed to the difference between ustilaginism and acrodynia as soon as my first articles were published.

Another close differential diagnosis of my ustilaginism is as against secalism, with which infant ustilago poisoning has admittedly a great symptomatological similarity, but has—at least pharmacognostically—an entirely different ætiology, just as "corn ergot" is an entirely different drug from *Secale cornutum.* "Corn ergot" is reported to have a similar, but not identical, effect as Secale. The pharmacologist Holste has *a priori* recognized in his Serbian toxicology (Beograd, 1930) the independent position of ustilaginism. The Serbian authors Barjaktarovic and Bogdanovic and Lewy and Tihomirov (Beograd, 1933) looked for the toxic effect of maize smut spores in a substance "which acted like ergotamine".

I am putting forward all this in order to corroborate my opinion that ustilaginism is an independent disease both (i) with regard to its clinical aspect in relation to pink disease (Swift) and to *acrodynia infantilis* (Feer), and (ii) with regard to its toxicological aspect in relation to secalism.

Neither did the French clinicians (Debré, Debré and Névot, Boucomont, Dechaume, Péhu) ever identify ustilaginism with *acrodynia infantilis.* Moreover Péhu and Boucomont differentiate precisely "real infantile acrodynia" from merely "acrodyniform syndromes of toxic ætiology".

I would like to add in this respect the names of a few more pædiatricians: Brdlik (Prague), Halbertsma (Holland), Reuss (Vienna) and Schachter (Marseilles). Dermatologists have also taken notice of the existence of an independent ustilago poisoning—Urbach, Wien (London) and Jadassohn (Switzerland). A synopsis on ustilaginism and on further clinical experiences in Zagreb of my collaborators was published by me in the *Progressus Medicinæ* (Istambul, 1948), which contains much more detailed information than this short communication can.

The misrepresentation of my views in English medical literature may have originated in inaccurate abstracting of my works and articles published in Serbo-Croat and German. I hope that this small contribution will help to clarify the position.

Summary.—The food poisoning of infants due to poisonous spores of *Ustilago maydis* (*"ustilagismus infantum"*), which the author first described in 1929-1930, has nothing in common with *acrodynia infantilis.* This sharp ætiological difference applies equally to the European form of acrodynia (type Feer) and, I believe, to the Australian form (pink disease, type Swift).

Yours, etc.,

E. MAYERHOFER,
Head of the Pædiatric Clinic,
Medical Faculty.

Zagreb,
Medvescar,
Yugoslavia.
July 17, 1950.

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MEDICAL PRACTICE IN 1872.

SIR: The following extract from the *New South Wales Medical Gazette* of February, 1873, has some interest in view of present discussions with friendly societies.

Medical Practitioners' Association.

At the commencement of the year 1872 the medical practitioners of Sydney interested in the working of benefit societies, seeing that they were not remunerated to the extent which their services were entitled to, and which the various benefit societies were able to afford, formed an association for the protection of their own interests, and at an early meeting the following rules were framed:

Rules of the Medical Practitioners' Association of New South Wales.

1. This Association shall consist of ordinary subscribing members and honorary non-subscribing members, both subject to the rules of the Association.

2. That the subscription of an ordinary member be one pound one shilling *per annum,* payable half-yearly.

3. That on and after March 1, 1872, the members of this Association shall not accept fresh appointments to benefit societies, unless in conformity with the following rules.

4. That appointments to benefit societies obtained before March 1, 1872, shall not be affected by the rules of the Association.

5. That the minimum rate of remuneration for attendance on members of benefit societies, their wives and families be 20s. per member *per annum,* exclusive of medicines. This rate is not to apply to societies consisting of women only.

6. That the minimum rate of remuneration for attendance on societies consisting only of widows and unmarried females shall be 16s. per member *per annum,* exclusive of medicines.

7. That the minimum fee for an accouchement shall be one pound eleven shillings and sixpence.

8. That abortions and premature confinements shall be paid for as ordinary accouchements.

9. That a fee of 2s. 6d. be paid for the examination of each candidate for admission to a benefit society.

10. All children of members will be entitled to attendance should their age not exceed sixteen years.

At the last meeting of the Medical Practitioners' Association it was decided that the following scale of charges might be substituted for Rules 5 and 6: For each visit in daytime at residence of patient, 3s. 6d.; advice at residence of medical attendant, 2s. 6d.; for each visit at night time, 7s. 6d.; that the night be deemed to commence at 8 p.m. and terminate at 8 a.m. It may be interesting to know that about twenty benefit societies have conformed to the rules of the Association. We recommend all young practitioners about to attend benefit societies in Sydney to join this Association.

Yours, etc.,

A. M. McINTOSH.

Sydney,
September 22, 1950.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED SEPTEMBER 16, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.
Ankylostomiasis	*	..	1	..	1	..	..	..	2
Anthrax	*	1(1)	..	..	..	..	..	..	1
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(2)	3(2)	2(2)	..	..	..	..	..	8
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(1)	..	..	..	..	..	2
Diphtheria	11(5)	1(1)	3(3)	..	1	..	..	..	16
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	..	..	..	..	..	..	..	..
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	*	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	1(1)	..	..	..	..	..	1
Measles	*	*	*	253(201)	*	..	1	1	254
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	10(6)	1	..	2(2)	2(2)	..	..	1	16
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	1	..	..	1
Rubella(*c*)	*	*	..	*	1(1)	*	..	2	3
Scarlet Fever	17(11)	20(11)	8(5)	3(3)	3(1)	1	..	..	52
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	17(14)	20(14)	16(2)	9(6)	16(12)	1	..	..	79
Typhoid Fever(*e*)	2	1	..	..	..	..	..	..	3
Typhus (Endemic)(*f*)	..	..	2	..	..	..	..	..	2
Undulant Fever	..	1	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	6	*	*	*	*	*	6
Whooping Cough	*	*	*	2(2)	*	*	..	..	2
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia,* Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

* Figures incomplete owing to absence of returns from the Northern Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Post-Graduate Work.

AUCKLAND UNIVERSITY COLLEGE, UNIVERSITY OF NEW ZEALAND.

DEPARTMENT OF POST-GRADUATE OBSTETRICS AND GYNÆCOLOGY.

Post-Graduate Course in Obstetrics and Gynæçology.

THE Auckland University College Council has decided to sponsor a comprehensive course of post-graduate obstetrics and gynæcology to be held in the Cornwall Hospital, Auckland, from March 5 to March 16, 1951. Contributions to the course will be made by the leading obstetricians and gynæcologists of New Zealand and also by physicians, surgeons, pathologists, hæmatologists and other specialists. It is expected that one or more specialists from overseas also will participate. It is believed that this course will afford an opportunity for graduates to learn of recent developments in obstetrics and gynæcology. There will be a limited amount of residential accommodation available, for which application should be made promptly. Further information can be obtained from Professor Sir Bernard Dawson, The Medical School, Dunedin, New Zealand, who has undertaken the organization and conduct of the course. A detailed programme will be published later, but in ample time prior to the above dates.

AUSTRALIAN SOCIETY OF ANÆSTHETISTS.

Short Course of Instruction in Practical Anæsthetic Methods.

A SHORT course of instruction in practical anæsthetic methods has been arranged for the week commencing Monday, October 30, 1950. It will be held in conjunction with the Royal Melbourne Hospital, the Alfred Hospital and the Children's Hospital. The fee for the course will be £2 2s. Application with remittance enclosed should be made not later than October 14, 1950, to Dr. L. Travers, Secretary, Australian Society of Anæsthetists, c.o. Department of Anæsthesia, the Royal Melbourne Hospital, Grattan Street, Parkville, N.2. A programme containing details of the course is available.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR NOVEMBER.

Demonstration at Flinders Naval Depot.

ON Wednesday, November 1, 1950, at 2.30 p.m., Dr. John Hayden will conduct a demonstration on "Modern Advances in Cardiology" at the Flinders Naval Depot, by arrangement with the Royal Australian Navy.

Week-End Course at Warrnambool.

A week-end course will be held at Warrnambool on November 18-19, 1950. The programme is as follows.

Saturday, November 18: 2.30 p.m., Dr. P. J. Parsons, "Jaundice"; 4 p.m., Dr. J. G. Hayden, "Hypertension"; 8.15 p.m., Dr. G. L. Grove, "Ano-Rectal Surgery".

Sunday, November 19: 10 a.m., Acting Professor J. W. Johnstone, "Hormones in Gynæcology".

The fee for this course is £2 2s., or 10s. 6d. per demonstration. Enrolments should be made with Dr. W. R. Angus, Koroit Street, Warrnambool. Telephone 52.

NOTE.—The place for this course, originally Mortlake, has been altered to Warrnambool.

Medical Appointments.

Dr. Edgar Herz has been appointed quarantine officer at Carnarvon, Western Australia.

Dr. Jessie E. J. Alexander has been appointed medical officer, Brisbane Mental Hospital, Goodna.

Dr. J. D. Sidey has been appointed Assistant Medical Superintendent, Surgical Branch, Royal Adelaide Hospital, Adelaide.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Schneeweiss, Joachim, M.B., B.S., 1950 (Univ. Sydney), Marrickville District Hospital, Marrickville.

Wilkinson, Thomas, M.B., 1950 (Univ. Sydney), Flat 2, 128 Johnston Street, Annandale.

Gillies, Douglas Neil, M.B., 1940, B.S., 1946 (Univ. Sydney), 26 William Street, Double Bay.

Diary for the Month.

OCT. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.
OCT. 13.—Queensland Branch, B.M.A.: Council Meeting.
OCT. 16.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
OCT. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

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